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SOME PROBLEMS IN ABNORMAL INTESTINAL PHYSIOLOGY ASSOCIATED WITH PERITONEAL ADHESIONS AND ILEUS

PANCOAST LECTURE*

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INTRODUCTION

THE invitation of the Philadelphia Roentgen Ray Society to give the Pancoast Lecture of 1945 is an honor which I deeply appreciate. It was not my privilege to know Dr. Pancoast well. As a youngster in radiology, I looked up to him as an outstanding leader. As the years have rolled on, his writings have stood out as important contributions. I am sure he would be interested in the problems about to be presented and in some speculative suggestions about possible explanations for them.

The purpose of this lecture is (1) to examine certain aspects of ileus and some influences which affect its development and course and (2) to discuss the local effect of adhesions on the small intestine.

The discussion must begin with certain phases of normal physiology of the small intestine which seem to have a bearing on the problems in abnormal physiology associated with ileus and peritoneal adhesions.

NORMAL PHYSIOLOGY OF THE SMALL INTESTINE

Movements of the small intestine involve three anatomically distinct and physiologically different structures, (1) the tunica muscularis, (2) the muscularis mucosae, and (3) the villi. The movement of the villi is not important in this discussion.

The following facts concerning the motor physiology of the tunica muscularis and the muscularis mucosae have been assembled from various standard text books and articles and have been dealt with in more detail elsewhere (Golden, 1945).

The *muscularis mucosae* is stimulated by the sympathetic portion of the autonomic nervous system but is not affected by the parasympathetic (vagus) portion. It moves independently of the tunica muscularis, and produces and changes the form of the mucosal folds.

Two types of movement of the *tunica muscularis* have been described, in each of which both the longitudinal and the circular layers take part:

* Pancoast Memorial Lecture, presented before the Philadelphia Roentgen Ray Society, November 1, 1945.

1. The myogenic or pendulum movements which in the dog are not affected by cocainization of the wall and which are, therefore, independent of nervous control.

2. The peristaltic movements which are disorganized by cocainization of the wall, and which are apparently controlled by reflexes coordinated through the intramural nervous system. Relaxation precedes and follows the peristaltic movement. Bayliss and Starling (1899) observed a blanching of the wall of the dog's intestine in a peristaltic constriction, indicating a temporary ischemia. As the earliest manifestation of fatigue they noted failure of relaxation. It is obvious that relaxation is just as important as contraction in a properly-coordinated peristaltic movement. Mechanical stimulation of the tunica muscularis by pinching caused a localized contraction (King and Arnold, 1922).

The chemical mediator theory of nerve action assumes that the characteristic effects of nerve stimulation are produced through the medium of diffusable substances produced at the end plates. Stimulation of the sympathetic nerves produces adrenalin (or sympathin, as Cannon called it) and affects "adreninergic" structures. Stimulation of the parasympathetic nerves produces acetylcholine and affects "cholinergic" structures.

Acetylcholine is rapidly destroyed by a specific enzyme, choline esterase, found only in nerve tissue. Choline esterase is inhibited or destroyed by certain drugs, e.g., prostigmine. Therefore, this drug prolongs and intensifies the action of acetylcholine and acts like a parasympathetic stimulator.

Adrenalin (sympathin) occurs in two forms, the inhibitor or I form, and the excitor or E form. Undifferentiated adrenalin, the I form, inhibits only cholinergic structures and stimulates adreninergic structures. The excitor or E form is partially oxidized adrenalin which has no inhibiting power but which stimulates cholinergic structures (Darrow). Oxidation products of adrenalin inhibit choline esterase in vitro (Waelsch and Rackow), and, therefore, resemble prostigmine; this observation suggests a possible mechanism for the above-described excitor effect of adrenalin E.

Much remains to be learned about the normal physiology of the intestine in spite of many years of investigation. The work of the neurophysiologists will be followed with great interest by those who have to deal with the gastrointestinal tract.

ILEUS

The word "ileus" is derived from a Greek word which means colic. Dictionaries define ileus as intestinal obstruction. Actually it is used in a much broader and more complicated sense. Ileus is usually used to mean a condition in which the intestine is dilated with the accumulation of gas and fluid and with other physiologic disturbances. Intestinal obstruction and ileus are two different things. Ileus may follow obstruction—mechanical ileus—or it may occur without obstruction—paralytic or, as Wangensteen calls it, functional ileus. Obstruction or, in other words, mechanical interference with the passage of intestinal contents, may be present without ileus.

Mechanical distention of the small intestine results in a number of profound disturbances both local and general, some of which are increased secretion, diminished absorption, failure to transport intestinal contents caudad (Ochsner), reflex inhibition of movement and tonus caudad and cephalad (Youmans, Meek and Herrin), narrowing and finally obliteration of intramural blood vessels which may result in gangrene and perforation (Storck and Ochsner), rise of blood potassium and specific gravity (Childs and Scudder).

The distention of the intestine with gas and fluid makes the detection of ileus easy by roentgen methods. The routine procedure is to take films with the patient lying on either side or even erect with the rays directed horizontally to show the fluid levels, in addition to a posteroanterior or an

anteroposterior projection. Evidence of ileus can be demonstrated earlier by this means than by any other method.

Distention by gas does not obliterate the mucosal folds of the jejunum which can be seen as fine cross striations in the gas shadow. The mucosal folds of the ileum are obliterated under the same conditions, resulting in smooth margins of its gas-distended loops. These facts make it usually easy to differentiate between gas-distended jejunum and ileum.

A discussion of the differential diagnosis between mechanical and paralytic ileus cannot be undertaken here.

Dilatation of the intestine and accumulation of fluid may occur without ileus. Moderate dilatation and accumulation of fluid occur in sprue, and without barium studies may be confusing (Kantor). In Figures 1*A*, 1*B* and 2 are illustrated two cases with dilated intestines proximal to a mechanical obstruction, yet the patients did not have ileus. These cases serve to emphasize the "other physiologic disturbances" in our concept of ileus. In these two cases the intestine was able to compensate for the obstruction because of the gradual development of the constriction, and the "other physiologic disturbances" did not occur. However, a point will be reached where physiologic compensation for the mechanical obstruction cannot continue and decompensation results. At this point the patient begins to have ileus. This apparently occurred in a seventy-year old business man who came to New York to attend a convention. After two or three days he suddenly developed severe abdominal pain and distention. At the hospital gas-distended loops of intestine with fluid levels were demonstrated. Barium injected through the Miller-Abbott tube after deflation disclosed a constriction in the terminal ileum, almost the duplicate of that shown in Figure 2. At operation carcinoma at the ileocecal junction was found with metastases in the liver. Obviously, the carcinomatous constriction had been developing for months. Why did the compensation of the intestine suddenly break? Possibly loss of sleep and fatigue associated

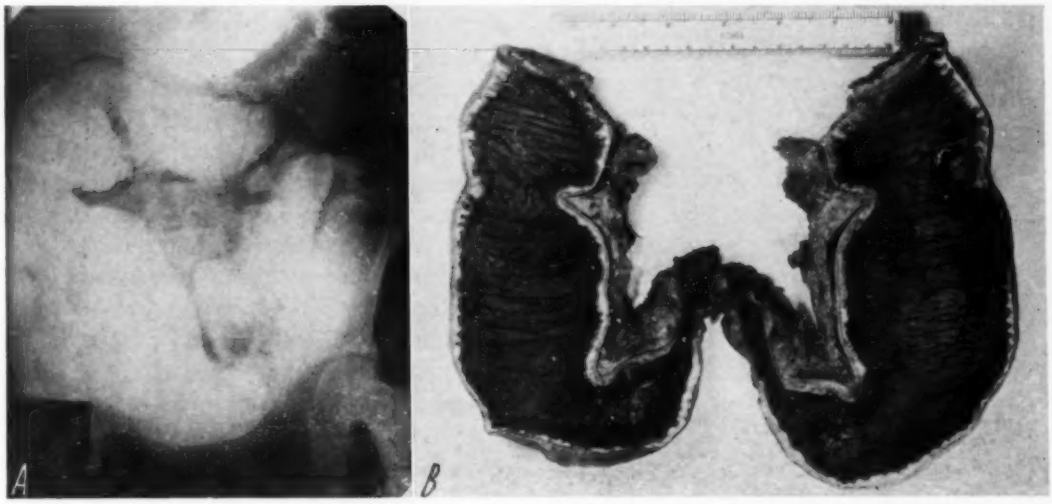

Fig. 1*A*. Dilatation of the jejunum proximal to a constriction resulting from regional enteritis of the lower jejunum. A twenty-six year old woman complained of recurrent attacks of diarrhea and cramping abdominal pain. The first attack occurred over three years before admission. She had none of the clinical signs of ileus. The intestine apparently compensated for the obstruction and dilatation.

Fig. 1*B*. Regional enteritis of the lower jejunum. The resected specimen shows apparently normal mucosal folds, hypertrophy of the wall and dilatation proximal to the slowly-developing constriction.

Fig. 2. Carcinoma of the terminal ileum with a narrow short constriction (arrow). Proximal to the constriction the intestine is dilated. A fifty-five year old woman complained of nausea and malaise for two months. She had no signs of ileus. (Courtesy of Dr. Harry M. Imboden.)

with attendance at a convention had something to do with it. Wangensteen postulates a plug of something, possibly undigested food, which may close a narrow passage and so precipitate ileus: this possibility cannot be denied. However, after deflation with the Miller-Abbott tube and the injection of barium we have never found complete obstruction.

The intestine, therefore, is vulnerable to rapid distention but seems to adapt itself very well to slowly-developing distention following gradually increasing obstruction.

Paralytic ileus may be initiated by a number of causes, three of which will be discussed.

Generalized purulent peritonitis is associated with gas distention of the intestine and fluid levels. Is this dilatation the result of an effect on the intramural nervous system or of an effect on the extramural sympathetic ganglia? In contrast, a constriction was produced by a localized abscess (Fig. 10).

Dilatation of the intestine may result from influences outside of and even remote from the intestine itself, e.g., pneumonia, injury of the spine, etc. A healthy seventeen-year old boy felt something give way in his back as he served at tennis but was able to finish the game. The following day the pain in the back increased steadily in severity, associated with increasing abdominal distention. By evening the pain was so severe that he was given morphine, without relief. During the following twelve hours he received a total of 90 mg. of morphine without effect on the pain. At the hospital the following morning he was given intravenous anesthesia so that he could be moved to the x-ray table. The films showed enormous distention of the stomach and intestines with gas but no abnormality of the spine could be seen. When the patient recovered consciousness from the anesthesia, the pain was much better. During the following thirty-six hours both the pain and the distention diminished and disappeared.

Hypoproteinemia may cause ileus. A man came to the hospital with severe burns over the face, neck, chest and arms. He received infusions of normal saline intravenously. The next day he began to have abdominal pain and on the third hospital day he had ileus. Deflation with the Miller-Abbott tube gave some relief. Hypoproteinemia was found. The saline infusions were stopped and transfusions of blood were given. As the blood protein returned to normal, the ileus disappeared.

Secondary paralytic ileus may complicate mechanical ileus. In some of our cases treated by deflation with the Miller-Abbott tube, redistention of the upper part of the intestine along the tube was detected after deflation had been apparently complete. When this is detected the tube should be pulled back until the tip lies in the upper part of the jejunum and the intestine deflated again. If this maneuver is not ac-

complished quickly enough the patient may die.

This secondary paralytic ileus is often associated with hypoproteinemia. Protein depletion often exists with gastrointestinal disorders, as Jones and Eaton pointed out in 1933. It may be aggravated by the inability of the patients with ileus to take food, by loss of protein through intestinal fluids withdrawn by the Miller-Abbott tube, or even induced by incorrect treatment. This is illustrated by the following case.

A fifty-one-year old man had diverticulitis in July, 1943. On September 14, 1943, he was admitted because of increasing abdominal pain. A film on September 15 disclosed the usual evidence of mechanical ileus. The Miller-Abbott tube entered the duodenum on September 17 and progressed slowly. Although the drainage was good he did not seem to do well. Beginning the second day after admission he was given 3,000 cc. of normal saline intravenously each day. On September 17 his temperature rose and remained elevated (101°–102° F.) until after the intravenous infusions had been discontinued. On September 20 the tube was arrested. Barium injected through the tube disclosed a kink (Fig. 3*A*) in the lower jejunum, which obviously represented the obstruction. The films also showed evidence of redistention of the jejunum along the tube. A film of the chest (Fig. 3*B*) at the same time disclosed heavy streaky shadows about both hila which were interpreted as pulmonary edema. The advisability of a blood protein determination was suggested. As no peripheral edema was present, his doctor preferred to believe that the lung shadows were due to pneumonia and gave him sulphadiazine. The blood protein was 5.1 which his doctor said was not low enough to be important. The Miller-Abbott tube was pulled back to the upper loop of jejunum. It again descended slowly, was arrested three days later, on September 23, and the kink was again demonstrated. Redistention of the jejunum along the tube was again present. Considerably more tube length could be seen at this time than was visible three days previously although the tip was arrested at the same kink in the lower jejunum. This suggests that the intestine was longer, probably because of hypotonicity of the longitudinal muscle. A film of the chest on the 23rd showed evidence of a considerable increase in the shadows around the hila and of fluid in the right hemithorax which had not been there three days previously. The blood protein on the 23rd was 4.9. The patient's doctor finally became convinced that he was dealing with hypoproteinemia associated with edema of the lungs and secondary paralytic ileus probably due to edema of the intestine. The infusions were stopped on the 24th and a liter of plasma was given. A film of the chest on September 27 disclosed no evidence of abnormality of the lungs. The patient received a high protein, high vitamin diet and made a rapid recovery. The Miller-Abbott tube was removed. Four weeks later, after he had gained weight and was in excellent condition, the tube was inserted for prophylactic purposes and he was operated upon. The kink was due to adhesions between the lower jejunum and the sigmoid resulting from the diverticulitis.

This case illustrates several important points. The excessive use of saline infusions is dangerous. In the presence of considerable salt, edema develops at higher levels of blood protein than without it. The first evidence of edema may appear in the lungs, as Jones and Eaton pointed out in 1933. The fever, which in another case reached 105° F., appears to be related to the pulmonary edema; the shadows in the lungs disappeared much too rapidly to be due to pneumonia. Redistention of the intestine may be taken as suggestive of hypoproteinemia although other nutritional factors, which cannot be discussed here, may play a part.

Discussion of Causes and Mechanism. These cases are examples of widely divergent causes of the distention of the intestine which we call ileus. The mechanical ob-

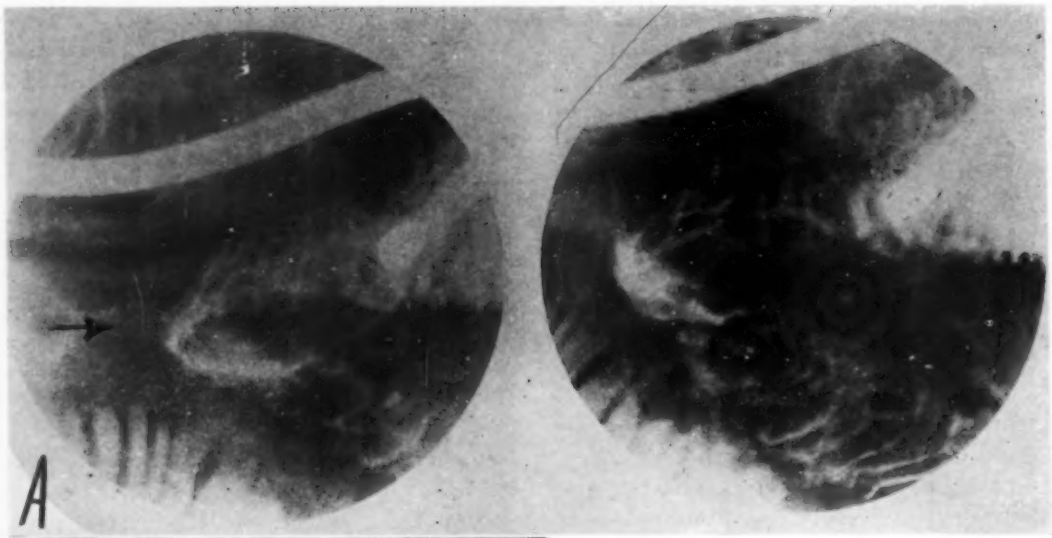

FIG. 3*A*. Kink in the lower jejunum which caused mechanical ileus later complicated by secondary paralytic ileus produced by hypoproteinemia. The kink was demonstrated by barium injected through the Miller-Abbott tube and was produced by adhesions between the ileum and sigmoid as a result of diverticulitis.

FIG. 3*B*. Edema of the lungs, caused by hypoproteinemia and excessive intravenous administration of normal saline solution. This film was taken at the time the kink (Fig. 3*A*) was demonstrated and re-distention of the jejunum along the Miller-Abbott tube was found. The blood protein was 5.1. No peripheral edema was present.

struction, the diffuse acute peritonitis, the wrenched back, and hypoproteinemia with edema of the intestine all appear to have exerted the same, or at least a similar, interference with intestinal physiology. How can the mechanisms set into motion by these different causes be correlated? Inasmuch as the parasympathetic nervous system is responsible for the tonus of the muscle coat, it seems reasonable to conclude that in some way the operation of the parasympathetic is interfered with. This might result from failure of the intramural nervous apparatus to produce acetylcholine in adequate quantities, as shown by Ingelfinger and Moss in sprue, or from too rapid destruction of acetylcholine by choline esterase, or, as suggested by Wangensteen, from over-activity of the sympathetic nervous system, the normal function of which is to inhibit the action of the parasympathetics. In the case of the boy with the wrenched back, the sympathetic mechanism would seem to be the most logical. Inasmuch as a localized infection (Fig. 10) caused a localized narrowing, the generalized dilatation of the intestine produced by a diffuse purulent peritoneal infection probably originates through some mechanism outside the intestinal wall. Von Gierke believes that splanchnic (sympathetic) stimulation occurs in rapidly spreading perito-

nitis; in peritonitis of longer duration the wall is damaged through over-distention, with toxic damage of the muscle and of the intestinal nervous apparatus.

In the hypoproteinemia cases, the interference seems to be associated with edema of the intestinal wall. Important information has come from the Departments of Radiology and Surgery at the Hospital of the University of Pennsylvania (Barden, Ravdin and Frazier, 1937) on disturbances in the function of gastrointestinal anastomoses due to edema. Leigh produced hypoproteinemia in dogs and by means of a balloon found that both tonus and peristalsis were depressed as edema developed. In clinical ileus Leigh noted slower progress of the Miller-Abbott tube when the patient had edema.

Edema of the intestine is localized largely in the submucosa. In the case shown in Figure 4 the blood protein was 5.0, of which 3.1 was albumen. The mechanism whereby this submucosal edema interferes with movement and other functions of the small intestine is not clear. Hodes (personal communication) found no appreciable disorder of the small intestine in several patients with beri-beri who had peripheral edema and who might be expected to have edema of the intestine. Dogs with hypoproteinemia (Barden, Ravdin and Frazier, 1937) induced by diet have disordered intestinal movements but do not get ileus. In both of these examples the development of edema must have been slow whereas the edema resulting from excessive fluid, salt and hypoproteinemia in ileus develops quickly. We have seen how the intestine can compensate for a gradually-increasing obstruction. It seems permissible to wonder whether with slowly developing edema the intestine succeeds in compensating in some way for the excessive fluid in its walls.

When the small intestine misbehaves in patients who have gastrointestinal disorders of any kind, hypoproteinemia must be suspected, bearing in mind that other nutritional disorders may play a part. One of our recent patients had a cholecystojejunostomy with end-to-side jejunojejunostomy for inoperable carcinoma of the head of the pancreas. A few days later the patient began to vomit. An examination of the upper part of the jejunum was requested because obstruction at the anastomosis was suspected. The jejunum was atonic, slightly dilated and very sluggish. The surgical resident, present at the examination, was asked about the level of the blood protein. He said that a determination would be done immediately. Later he reported that the test showed the blood protein to be "way down" and that measures to correct the hypoproteinemia were immediately instituted. However, that night the patient disrupted his wound, an event usually associated with hypoproteinemia.

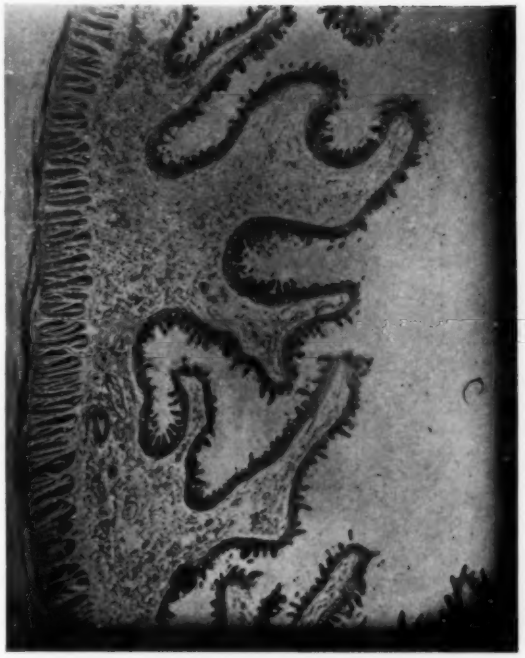

Fig. 4. Edema of the intestine due to hypoproteinemia. The total blood protein was 5.0, albumen 3.1 The fluid is collected in the submucosa, not within the mucous membrane and has produced widening of some of the folds. The separation of the bundles of circular muscle is due to artefact, not to edema. (Courtesy of Dr. Edith Sproul.)

The problem of fluid, salt and blood protein regulation is complex and I am not qualified to discuss it.

Fig. 5. Segmentation with immobility of the small intestinal loops (four hours after the ingestion of barium), associated with multiple peritoneal adhesions following generalized peritonitis from acute appendicitis in a ten-year old girl. This appearance was called the "irritation pattern" by Soper. The segmentation is caused by failure of short segments of intestine to relax. Barium did not reach the cecum in four hours.

PERITONEAL ADHESIONS

Soper (1929) showed that multiple peritoneal adhesions were associated with a disturbance in the motor function of the small intestine which produced what he called the "irritation pattern." It is characterized by multiple constrictions in the barium shadows (Fig. 5).

The commonest source of adhesions which cause trouble is said to be operation for appendicitis and the next commonest is operation on the female pelvic organs. In my experience the latter is the more common.

Whatever may be the source, a knuckle or kink in the intestine can almost always be demonstrated after deflation with the Miller-Abbott tube. A typical kink is shown in Figure 6. Proximal to the kink the lumen is slightly wider than normal. Distal to the kink the lumen is approximately normal in caliber. Both limbs of the kink are narrow but the narrowing is more extensive in the distal than in the proximal limb. The kink was produced by a band of adhesions described by the surgeon as one inch in width but the narrowing shown on the film is almost twice that long. The significance of these facts will be discussed later.

Adhesions may cause pain without

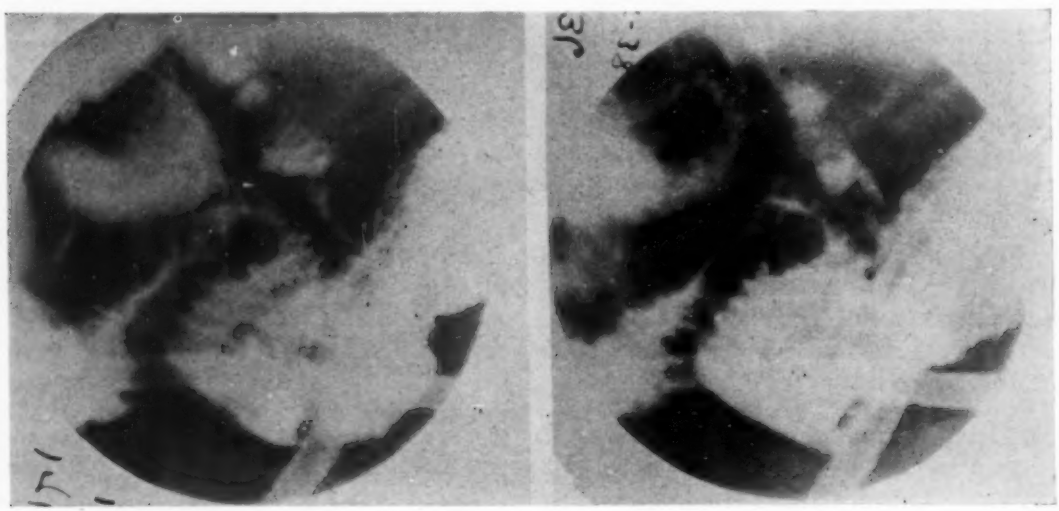

Fig. 6. A typical knuckle or kink in the small intestine uncovered by the pressure cone after deflation and the injection of barium through the Miller-Abbott tube. The adhesion in a fifty-eight year old woman followed hysterectomy; it was only one inch in length and bound the two loops together. Both limbs of the kink are narrow but the narrowing is more extensive in the distal limb.

producing ileus. In some cases the Miller-Abbott tube can be used to demonstrate and to localize with certainty the offending kink. The balloon distended with 20 cc. of air may be arrested by a knuckle which might be difficult or impossible to detect by the usual barium study of the small intestine, particularly if the knuckle lies low in the pelvis. In the case illustrated in Figure 7, the narrowing is in the distal limb. Although other adhesions were present, release of the adhesion which produced the kink was followed by relief of pain.

Not all adhesions which cause pain produce kinks or knuckles sufficiently sharp to arrest the balloon of the Miller-Abbott tube, although in our experience many of them do.

Scarring of the intestinal wall due to endometrioma produced two knuckles with narrowing. The narrowing shown by the barium shadow (Fig. 8*A*) is considerably longer than the sharp narrow indentations shown on the resected specimen (Fig. 8*B*). The narrowing in the barium shadow, therefore, could hardly be due to fibrotic constriction but must result from spasm of the muscle in the region of the lesion.

Fig. 7. A knuckle of ileum caused by adhesions following hysterectomy in a thirty-year old woman. The narrowing (arrow) is in the distal limb of the kink. The patient had repeated attacks of abdominal pain without ileus. The balloon of the Miller-Abbott tube was arrested at the site of this knuckle, which was then demonstrated by injecting barium through the tube. Release of the adhesion binding the two limbs of this loop together was followed by relief of pain.

In another case the terminal ileum was

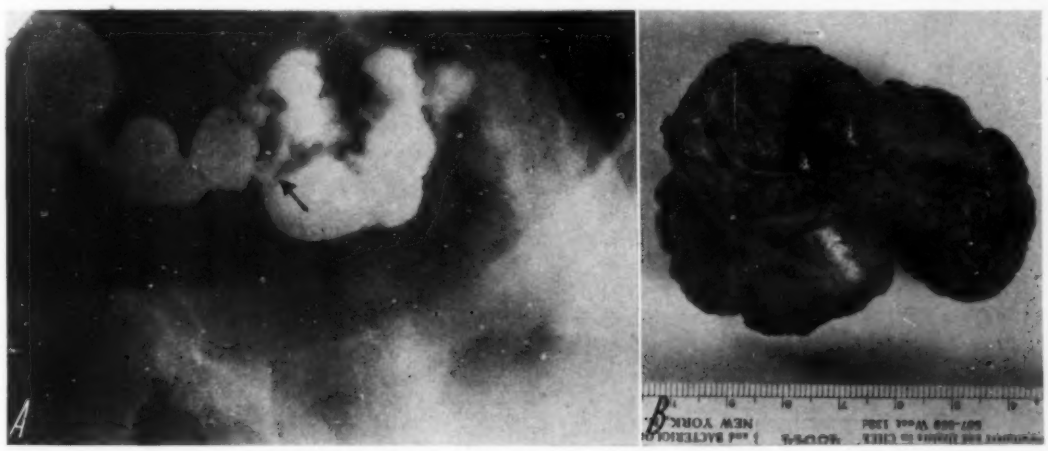

Fig. 8*A*. Two narrow knuckles in the ileum (arrows) produced by scarring due to endometrioma of the wall. The narrowing of the lumen of these two segments demonstrated by the barium is considerably longer than the very sharp indentations seen on the external surface of the resected specimen (Fig. 8*B*). At the fluoroscopic examination barium was propelled through the knuckles without expanding the lumen.

Fig. 8*B*. Endometrioma of the wall of the ileum. A photograph of the resected specimen shows that the actual indentations produced by the scarring of the wall were very sharp and narrow. The narrowed lumen of the two segments disclosed by the barium shadows (Fig. 8*A*) must have been the result of spasm at the site of the fibrosis in the wall produced by the intramural tumor. (Courtesy of Dr. A. Purdy Stout.)

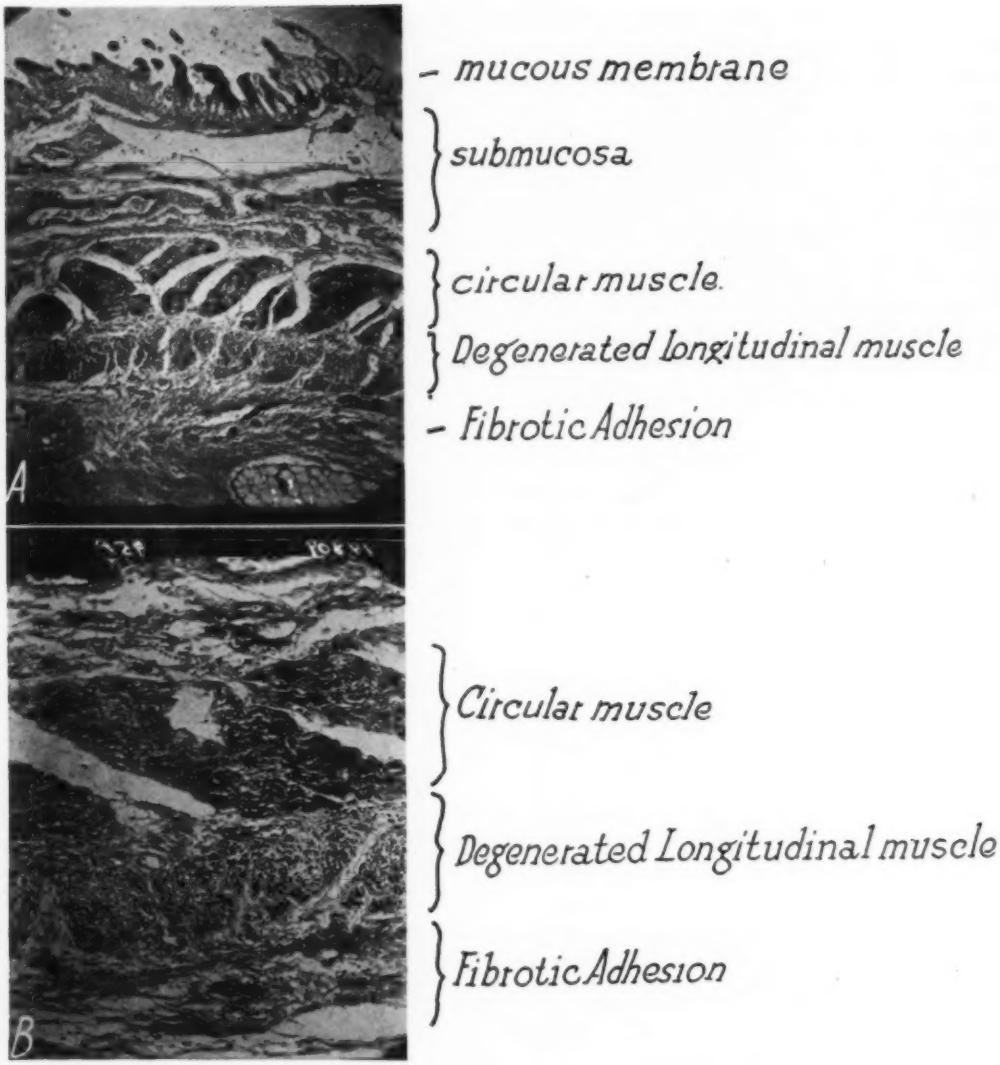

Fig. 9*A*. A low-power photomicrograph of the intestinal wall at the site of an old adhesion which caused a knuckle. The intestine was slightly narrowed at the knuckle. The fibrotic adhesion is at the bottom of the photomicrograph. The fibrosis extends through the longitudinal muscle and involves the plane between the two layers where the mysenteric ganglia of Auerbach lie. The circular layer is involved very slightly. Evidence of degeneration of the muscle cells of the longitudinal layer can be seen (see Fig. 9*B*).

Fig. 9*B*. A higher power photomicrograph shows better the degeneration of the fibrotic longitudinal muscle and the relatively normal circular muscle. (Courtesty of Dr. Edith Sproul.)

adherent to the right cornua of the uterus following operation for ectopic pregnancy with removal of the right tube and ovary. For twenty-five years after this operation the patient was plagued by attacks of right lower quadrant pain. A narrowed area was demonstrated by barium studies in the terminal ileum. At operation the ileum at the site of attachment to the uterus was narrower than on either side of the attachment, although the adhesion involved only the margin of the intestine adjacent to the uterus and did not pass around the wall.

Discussion. Textbooks of surgery and surgical pathology attribute the intestinal obstruction produced by adhesions to mechanical pressure, traction, kinking or torsion. The observations on the cases just

described indicate that the effect of adhesions on the wall of the intestine is a little more complex than mere mechanical pressure. The narrowing at the site of the adhesions must be due to a contraction of the circular muscle and not to pressure from the outside. In meditating on this abnormal phenomenon a number of questions come to mind.

1. How deeply into the intestinal wall does the inflammatory process extend?

The answer could not be found in textbooks. When asked about it, a number of pathologists stated that they had never investigated this question carefully but had the impression the wall was involved only superficially. Dr. Edith Sproul, Assistant Professor of Pathology, kindly made some sections of the intestinal wall at the site of a peritoneal adhesion found at autopsy. The adhesion produced a knuckle by binding two loops together. The distal limb of the kink was narrower at autopsy than the proximal limb. The section was taken directly under the adhesion. The photomicrographs (Fig. 9) show that the inflammatory process extended through the outer or longitudinal layer of muscle. The region of Auerbach's plexus between the two layers is fibrosed but the inner or circular layer is involved very little, if at all. The muscle cells of the outer longitudinal layer show evidence of degeneration. Unfortunately, the section did not extend clear around the entire circumference of the wall. Similar studies of many cases must be done before conclusions can be drawn as to the pathological changes in the wall of the intestine at the site of peritoneal adhesions.

2. Does the acute inflammation or the fibrosis resulting from it produce a localized disturbance in the motor physiology of the intestine?

Figure 10 shows a narrowing of the ileum at the site of a localized abscess resulting from appendicitis. The contrast between the effect of this localized inflammation and the effect of a generalized peritonitis was commented upon above. Evidence was presented previously that the narrowing of the lumen at the site of an adhesion cannot be attributed to purely mechanical pressure. Since release of the kink by severing the adhesion is usually followed by relief, it would seem that the fibrosis within the wall is probably not in itself responsible for pain, because the severing of the adhesion outside would not affect the fibrosis within the wall. However, in some cases the intramural fibrosis may be of importance in this respect. Consequently, it seems that both a localized acute inflammation and a fibrotic process in the wall at the site of an adhesion are capable of producing a localized disturbance in the motor physiology of the intestine, manifested by narrowing.

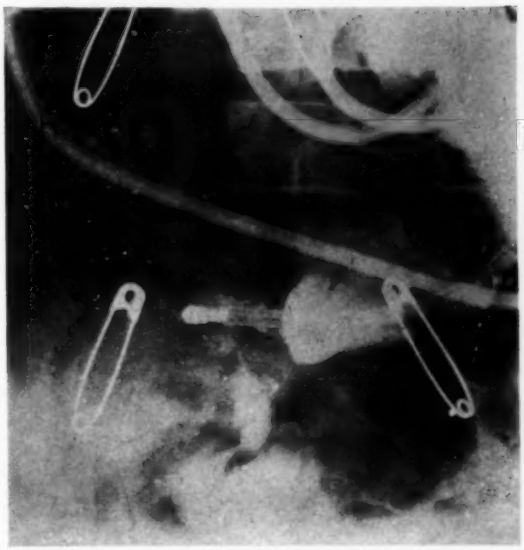

Fig. 10. Narrowing of the ileum at the site of a localized abscess following appendicitis. The patient had mechanical ileus. After deflation by and arrest of the Miller-Abbott tube, the narrowed kink was disclosed by barium injected through the tube. This localized narrowing at the site of an abscess is in striking contrast to the distention of the intestine (paralytic ileus) caused by a diffuse generalized peritonitis.

3. Is mechanical stimulation produced by peristaltic movements and transmitted caudad through the adhesions to the wall lower down necessary to produce the narrowing?

Fig. 11. Elongated narrowing of a segment of ileum associated with strangulation. This loop was caught under a shelf of adhesions. The patient, a fifty-nine year old woman, was admitted with mechanical ileus. After deflation with the Miller-Abbott tube, the narrowed loop was demonstrated by the injection of barium through the tube.

4. Why does the narrowing occur largely in the distal limb of the kink?

These two questions must be considered together. Inasmuch as mechanical stimulation by pinching causes a contraction, it would seem possible that a pull by an adhesion inserted into the wall could do the same thing. The peristaltic wave is associated with a contraction of the longitudinal as well as of the circular muscle. The pull of the longitudinal muscle would be transmitted ahead of the wave by the adhesion. This would be an explanation for the more intensive narrowing of the distal than of the proximal limb. Furthermore, under normal conditions a reflex relaxation (inhibition) of both the longitudinal and the circular muscle precedes the peristaltic contraction. It seems quite possible that a mechanical stimulus, transmitted to the wall ahead of the contraction wave, could interfere locally with relaxation and with the orderly operation of the peristaltic reflex and thus disturb the propulsion of intestinal contents.

5. Does the mechanical stimulus affect the muscle directly, producing a contraction of myogenic origin, or does the stimulus operate through the colinergic intramural nervous system?

This question probably cannot be answered. Nachmansohn (personal communication) says that any stimulus, either mechanical or electrical, would result in the production of acetylcholine. This would amount to an effect developed through the intramural nervous system. However, both mechanisms may play a part.

6. Why does the narrowed area fail to expand when the stimulus is not active?

7. Does the failure to relax result from absence of a sympathetic effect on the tunica muscularis, from local failure of choline esterase to destroy acetylcholine, or from diminished local oxygen supply in the muscle?

Little evidence is at hand upon which an answer to these questions could be based. Bayliss and Starling observed blanching of the wall under a peristaltic wave in the dog's intestine. At operation the wall of the human stomach becomes pale under a peristaltic wave. If the mechanical stimulus is repeated frequently enough so as to prevent relaxation, is it possible that local interference with and reduction of the blood supply to the muscle may result? The long narrowed segment of ileum in Figure 11 which produced mechanical ileus was strangulated. Dr. Edith Sproul (personal communication) has observed a number of cases at autopsy in which adhesions involving the intestinal wall were associated with localized narrowing which persisted after death. Is it possible that prolonged or repeated ischemia may result in fibrosis of the muscle?

Twenty-five or thirty years ago deep incisuras in the stomach opposite a lesser curvature ulcer were not infrequently seen, but are rarely encountered now. This resulted in the B type of hourglass stomach.

The incisura presumably began as spasm but later the contracted segment of muscle became fibrotic and the constriction in the stomach became permanent. Why did the muscle change to fibrous tissue? Blanching of the stomach wall under a peristaltic wave has already been mentioned. Prolonged spasm of a narrow segment might well result in persistent ischemia. The normal result of reduced blood supply, and therefore of oxygen supply, is for cells which have a higher oxygen requirement like muscle to be replaced by cells with a lower oxygen requirement. It is an accepted principle in pathology that ischemia leads to fibrosis. This might explain the development of a permanent incisura in the stomach.

How far this analogy will fit the effect of adhesions on the wall of the intestine is questionable. Unfortunately no information is available concerning the condition of the muscle in the contracted areas of the intestine at the site of adhesions. In the single case described above (Fig. 9), the circular muscle was not fibrosed and therefore showed no effect attributable to persistent ischemia. Study of a large series of similar cases is highly desirable. It is important to determine how far the intramural fibrotic process extends around the wall beyond the externally visible attachment of the adhesion. Although the exact mechanism cannot be determined now, the failure of the narrowed area to expand in life and the persistence of narrowing after death must be the result of some abnormality within the wall not limited to the exact extent of the adhesion.

The blanching of the wall under a peristaltic wave in the dog's intestine indicates that temporary ischemia is normally associated with a peristaltic contraction. May not a similar diminution in blood supply, perhaps a lesser degree of it, accompany the persistent abnormal constrictions shown in Figures 6, 7 and 8? If a peristaltic contraction is superimposed, further reduction in blood supply would occur temporarily. Ischemia of the heart muscle in coronary insufficiency and ischemia of the skeletal muscle associated with narrowing of the peripheral arteries is associated with pain. Under those conditions it has been quite well established that the pain is induced by lack of oxygen in the tissues because the pain can be relieved by measures which increase the oxygen supply in the muscle or which decrease the need for oxygen. The pain associated with peritoneal adhesions without ileus is usually cramping in character and is increased after eating. It is usually attributed to peristaltic pull of the adhesions on the sensitive peritoneum. Is it possible that ischemia in the intestinal wall at the site of the adhesion may have something to do with the pain?

SUMMARY

In the Pancoast Lecture of 1945 we have discussed the significance of certain observations on the small intestine in cases of peritoneal adhesions, obstruction and ileus. On the background of present day concepts of intestinal physiology, we have speculated on possible explanations for some of these abnormal phenomena.

Ileus is more than mere dilatation of the intestine. As ordinarily used, this term includes far-reaching disorders both within and outside of the intestinal wall.

The intestine is highly vulnerable to acute distention, but shows a remarkable ability to compensate for a gradually-developing obstruction with slow dilatation.

Hypoproteinemia and edema are frequent complications of mechanical ileus and may be responsible for a superimposed paralytic ileus.

The trouble caused by peritoneal adhesions appears to be more than the effect of mere mechanical pressure or angulation. A localized disturbance in the motor physiology of the intestine may occur at the site of an adhesion. This is manifested by narrowing of the lumen which may persist after death. The mechanism by which this is produced is not clear. Exact

knowledge concerning the histologic changes within the wall at the site of adhesions is not available. It seems possible that a mechanical stimulus from the pull of the peristaltic contraction transmitted caudad through the adhesions to the wall ahead of the peristaltic wave may play a part in causing the localized narrowing.

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A PULMONARY REACTION FOLLOWING INTRA-BRONCHIAL INSTILLATION OF LIPIODOL IN BRONCHIAL ASTHMA

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EXPERIENCE has shown that the use of iodized oil in the bronchopulmonary tree may be accompanied by some danger. This has been pointed out by Amberson and Riggins,[1] who believe that a slight exudative reaction usually occurs about deposits of lipoidol in the healthy lung. They do not consider this occurrence to be harmful in the clinical sense. These authors suggest that a transudation of edema fluid may occur in considerable amount. This, they believe, may account for the rapidly developing roentgenographic lobar opacity reported by some authors following intrabronchial instillation of iodized oil.

Sensitivity to lipoidol or its separate ingredients, however, has not been emphasized as a cause of pulmonary reaction following bronchography. Anderson[2] has reported on the treatment of 100 cases of bronchial asthma by intratracheal injections of lipiodol. This author mentions noting instances of certain untoward reactions following this procedure which were characterized by chest pain and temperature. Sheldon[3] has reported a case of an acute pulmonary episode following instillation of lipiodol in an individual with a known allergy to iodides. A fatality has been reported by Macdonald[4] following bronchography with lipiodol. This was the case of a thirteen year old girl who experienced nausea and vomiting after the lipiodol injection, became cyanotic, and died in twenty hours. This child had suffered from asthmatic attacks for many years and sensitization tests, carefully carried out, indicated that she was hypersensitive to several types of allergens. Macdonald believed that her death was due to an allergic reaction to lipiodol.

The importance of sensitivity to contrast media was recently reviewed comprehensively by Pendergrass, *et al.*[5] who reported on a survey of unfavorable sequelae and deaths following the intravenous administration of contrast media. Although these studies were concerned principally with those contrast media in use in genitourinary diagnosis, such as diodrast, diodrast compound, iopax, neo-iopax, and skiodan, certain of these observations are pertinent to the present report. These authors postulate that the unfavorable sequelae found may be due to hypersensitivity, and that the incidence of such reactions may be greater in allergic individuals. Accordingly they point out the necessity of taking a thorough history as to personal or family allergic background, as well as performing adequate sensitivity tests in all individuals before the use of iodine-containing contrast media.

In a recent study of 40 cases of bronchial asthma, bronchograms were done as a part of a clinical investigation, using lipiodol as the contrast medium. In 3 of the cases, an acute febrile illness developed following the introduction of the medium associated with roentgen evidence of pulmonary infiltration, and certain features noted in these cases are considered worthy of reporting.

METHOD

The lipiodol used in these cases was the commercial product, consisting of iodized poppy seed oil (40 per cent), an iodide addition product of poppy seed oil containing 39 to 41 per cent of iodine (0.54 gm. of iodine per cc.) in organic combination.[6]

Butyn sulfate 2 per cent was introduced intranasally, and anesthesia of the tracheobronchial tree was attained by the gravity method. Lipiodol was used to visualize both lower lobes and the right middle lobe. No catheter was used to instill the lipiodol.

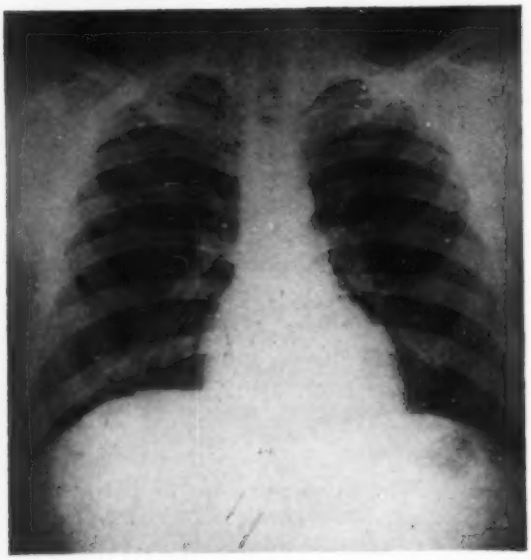

FIG. 1. Case 1. Routine posteroanterior roentgenogram of the chest, July 7, 1943. Negative for pulmonary pathology.

REPORT OF CASES

CASE 1. A white male, aged twenty, was admitted to the hospital July 6, 1943, with the chief complaint of wheezing and a cough. He had been a college student in civilian life, and gave a family history of two sisters and one nephew having hay fever and asthma. He had hives in childhood, his last attack occurring at the age of twelve. In August, 1941, he began to sneeze and had an acute rhinitis, with redness and watering of the eyes, cough, and tightness of the chest. These attacks lasted until the late fall. About August 1, 1942, one year later, his symptoms recurred. He entered the armed services in December, 1942. In April, 1943, he began to have wheezing which interfered with his military duties so severely that he had to be hospitalized. On admission to the hospital on July 6, 1943, many sibilant râles were heard throughout both lungs, with a prolonged expiratory phase of respiration. The remainder of the physical examination was normal. The temperature was 98.6° F. Laboratory examination revealed the Kahn test, urine, blood calcium, sedimentation rate, and gastric analysis to be within normal limits. The complete blood count was as follows: red blood cells 6.36 million; hemoglobin 19 gm.; white blood cells, 13,050; the differential was as follows: eosinophiles 20 per cent, stab cells 2 per cent; segmented cells 34 per cent, lymphocytes 40 per cent, and monocytes 4 per cent.

Routine roentgenography of the chest performed on July 7, 1943, was negative for pulmonary pathology (Fig. 1).

On July 20, 1943, a bronchography was performed, the lipiodol being instilled into both lower lobes and the right middle lobe. Bronchograms of the chest following lipiodol instillation revealed a normal bronchial tree which was well outlined by the lipiodol (Fig. 2).

Nine days later on July 29, 1943, the patient complained of malaise and cough. His temperature rose to 101° F. Physical examination at this time revealed coarse moist râles at the bases of both lower lobes. The white blood count was 15,300, with the following differential: juvenile cells 1 per cent, stab cells 2 per cent, segmented cells 76 per cent, lymphocytes 21 per cent. A roentgenogram of the chest on the following day revealed a hazy density of both lower lung fields, most marked at the left base, along the cardiac border. There was also an area of patchy density in the right upper lung field (Fig. 3). An extensive urticarial rash appeared for the first time, covering both upper arms and shoulders. The urticarial lesions disappeared after twenty-four hours. His temperature rose to 102.4° F. and sulfathiazole by mouth was begun the same day. A total of 22 gm. of sulfathiazole by mouth was administered

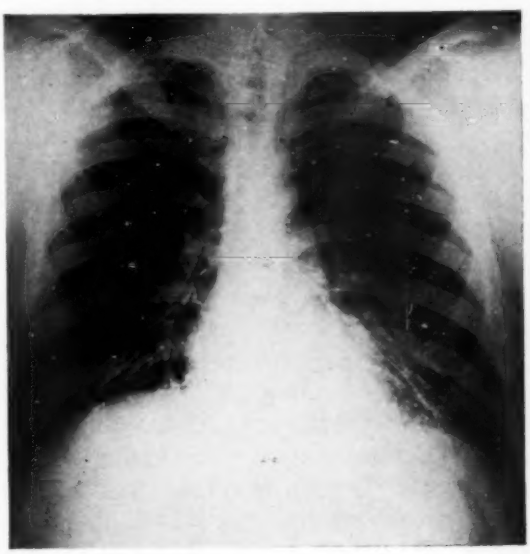

FIG. 2. Case 1. Bronchogram taken July 20, 1943, with filling of both lower lobes and the right middle lobe. Normal bronchial tree.

in the next three days. The symptoms rapidly subsided, and the temperature returned to normal after forty-eight hours. The physical signs of coarse moist râles persisted at the bases of both lower lobes and did not completely disappear until one week following the onset of the illness.

Routine roentgenograms of the chest were taken on August 6, 1943, which showed almost complete resolution of the pulmonic densities in both lung fields (Fig. 4).

Comment. This patient had a family history of hay fever and asthma. He was hospitalized because of bronchial asthma. Nine days after a normal bronchogram, he developed an acute episode characterized by a rise in temperature, cough, expectoration and malaise. An urticarial eruption appeared with the onset of this acute illness and disappeared in forty-eight hours. No eosinophilia was found. The white blood cell count was elevated. Roentgen examination of the lungs revealed bilateral basal densities, more marked on the left side. An area of patchy density was also present in the right upper lung field. The roentgen appearance suggested the presence of pneumonitis. These densities conformed to the areas of distribution of the lipiodol, except for the density in the upper right lung field. One may conjecture as to the possibility of lipiodol having been distributed to this area by coughing.

Fig. 3. Case 1. Posteroanterior roentgenogram of the chest, July 30, 1943, ten days following bronchography. Areas of increased density at both bases, more marked at the left cardiophrenic angle. Patchy area of increased density in the upper right lung field, near the upper axillary region.

Fig. 4. Case 1. Posteroanterior roentgenogram of the chest, August 6, 1943. Almost complete disappearance of areas of increased density. Patchy infiltration still present at left base and in right upper lung field.

Case II. A white male, aged twenty-one, was admitted to the hospital August 11, 1943, with the chief complaint of paroxysmal nocturnal dyspnea. In civilian life he had been a plastic moulder. A cousin suffered from asthma. He knew of no other member of the family with an allergic condition.

This patient had had dyspnea and fatigue for many years. He had been unable to engage in athletics. For two years previous to his admission to the hospital, he had noted frequent attacks of sneezing and coughing at night. He was inducted into the Army on January 28, 1943. For four months he had had paroxysmal nocturnal dyspnea, relieved by sitting upright in bed and he was hospitalized for this reason.

On admission to the hospital on August 11, 1943, physical examination revealed generalized sonorous râles throughout both lungs. Sedimentation rate, urine analysis, Kahn test, blood calcium, basal metabolic rate, gastric analysis, and stool examinations were all within normal limits. The complete blood count on August 12,

1943, showed 5.87 million red blood cells, 16.5 gm. hemoglobin, and 10,150 white blood cells, with the following differential: eosinophiles 16 per cent, stab cells 4 per cent, segmented cells 51 per cent, lymphocytes 27 per cent, and monocytes 2 per cent. A blood count taken on August 23, 1943, revealed 11,750 white blood cells with the following differential: eosinophiles 4 per cent, segmented cells 74 per cent, lymphocytes 22 per cent. Roentgenograms of the sinuses showed a pansinusitis. A routine roentgenogram of the chest taken on August 12, 1943, showed no pulmonary pathology.

Bronchography was performed on September 9, 1943, the lipiodol being instilled into both lower lobes and the right middle lobe. Bronchograms of the chest following this procedure revealed a normal bronchial tree which was well outlined by the lipiodol.

Twelve days later, on September 21, 1943, the temperature rose to 99° F. The following day, the patient complained of malaise and developed a persistent cough. The temperature continued at a level of 99.8° F. On the next day, September 23, 1943, crepitant râles were discovered for the first time at both bases, and his lips appeared cyanotic. The patient did not appear seriously ill. He raised sputum but it contained no blood. The white blood count was 22,800 with the following differential: eosinophiles 22 per cent, stab cells 5 per cent, segmented cells 50 per cent, lymphocytes 17 per cent, and monocytes 6 per cent. A roentgenogram taken on this day revealed hazy densities in both lower lung fields, more marked at the right base, where the density occupied the lower one-third of the lung field. The density at the left base was less extensive and was present at the level of the fifth and sixth ribs anteriorly, adjacent to the cardiac border.

Sulfadiazine therapy was instituted, and a total of 14 gm. was given in the next two days. On September 24, the white blood cell count fell to 18,100 with the following differential: eosinophiles 18 per cent, stab cells 7 per cent, segmented cells 54 per cent, lymphocytes 20 per cent, and monocytes 1 per cent. The following day 15,000 white blood cells were found, with the following differential: eosinophiles 27 per cent, basophiles 1 per cent, stab cells 4 per cent, segmented cells 45 per cent, lymphocytes 18 per cent, and monocytes 5 per cent. Subsequent roentgenograms taken on September 25 and 29, showed a gradual resolution of the pneumonic densities in both lung fields. A blood count taken on the latter date showed 8,000 white blood cells, with the following differential: eosinophiles 9 per cent, segmented cells 52 per cent, lymphocytes 30 per cent, and monocytes 4 per cent. A roentgenogram taken on October 4, 1943, thirteen days following the onset of this illness, showed complete resolution in both lung fields, with residual lipiodol still present at the extreme right base and in the area in the left lung where the pulmonic density had been present.

Comment. This patient had bronchial asthma. Twelve days following a normal bronchogram, he developed an acute pulmonary episode characterized by cough, expectoration and fever. Roentgen examination of the lungs showed bilateral basal densities. A marked eosinophilia was discovered which paralleled the course of the acute illness.

In view of the extensive bilateral pneumonic densities found on roentgen examination, the temperature curve was startingly benign. The highest temperature noted was 99.8° F., the patient in general felt quite comfortable throughout his illness, in spite of the persistence of physical signs in both lower lobes for more than a week. It was the clinical impression that the sulfadiazine had not had any particular effect on the course of the illness.

CASE III. A white male, aged thirty, was admitted to the hospital September 2, 1943, complaining of tightness in the chest and dyspnea. He had been a musician in civilian life. He stated that his mother suffered from asthma, and that he had hay fever and asthma for twenty years. He had noted an increase in the severity of his complaints during the preceding two years. He gave a long history of sensitivity to pollens and dust, and frequent attempts to desensitize had given no relief. He stated that he had never had hives. He was inducted into the Army on March 11, 1943, and was hospitalized on September 2, 1943, because of an exacerbation of his symptoms. Physical examination revealed sibilant râles heard throughout both lung fields. The sedimentation rate, Kahn, Wassermann, blood calcium, urine, gastric analysis, and basal metabolic rate were within

normal limits. A complete blood count revealed 4.7 million red blood cells, 16.2 gm. hemoglobin, and 7,050 white blood cells, with the following differential: eosinophiles 13 per cent, stab cells 2 per cent, segmented cells 67 per cent, and lymphocytes 28 per cent. A routine roentgenogram of the chest taken September 4, 1943, was negative for pulmonary pathology (Fig. 5).

Bronchography was performed September 16, 1943, with instillation of lipiodol into both lower lobes and the right middle lobe and revealed a normal bronchial tree (Fig. 6).

Two days following the bronchogram, the patient noted a sudden urticarial eruption covering the trunk and upper extremities which

Fig. 5. Case III. Routine posteroanterior roentgenogram of the chest, September 4, 1943. Negative for pulmonary pathology.

itched markedly. This eruption disappeared within forty-eight hours. On September 24, 1943, eight days following the bronchogram, he developed a tightness in the chest which was followed the next day by chilly sensations, malaise, substernal pain, and cough. Physical examination revealed coarse moist râles over both lower lobes, in addition to the generalized sibilant râles found on previous examinations. The white blood cell count rose to 13,800 with the following differential: eosinophiles 1 per cent, juvenile cells 5 per cent, stab cells 10 per cent, segmented cells 61 per cent, lymphocytes 19 per cent, and monocytes 4 per cent. On September 25 his temperature rose to 100° F.

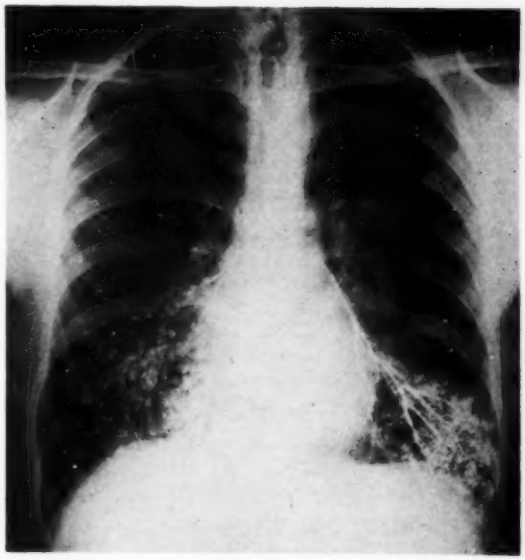

Fig. 6. Case III. Bronchogram taken September 16, 1943, with filling of both lower lobes and the right middle lobe. Normal bronchial tree.

and fell to normal on the following day. A roentgenogram taken at this time, on September 26, 1943, revealed densities of both lower lung fields which had a cotton-like appearance The density on the left side was more marked

Fig. 7. Case III. Posteroanterior roentgenogram of the chest, September 26, 1943, ten days following bronchography. Areas of increased density in both lower lung fields. The density at the left base is pneumonic in character.

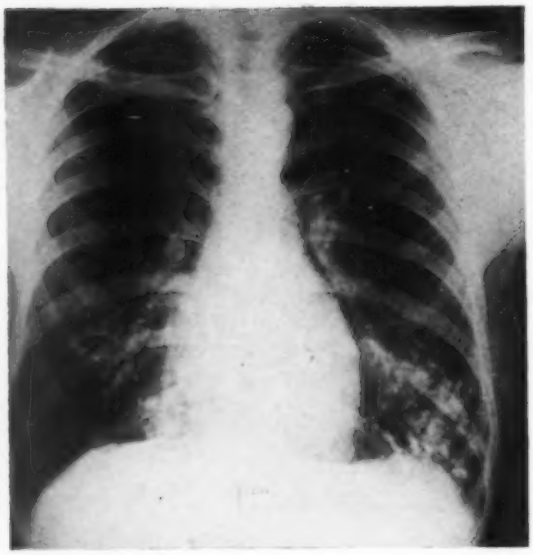

FIG. 8. Case III. Posteroanterior roentgenogram of the chest, October 4, 1943. Marked resolution of the densities seen in Figure 7. Residual lipiodol is still present in both lower lung fields.

and was pneumonic in character (Fig. 7). No sulfa drugs were given because of the experience in the two previous cases. Except for the initial temperature rise to 100° F., the patient remained afebrile and there was a complete disappearance of abnormal physical signs in both lungs after one week. A routine roentgenogram taken October 4, 1943, nine days after the onset of the illness, showed almost complete resolution of the pulmonary densities with residual lipiodol still seen in both lower lung fields (Fig. 8). A blood count taken on this day showed 8,500 white blood cells, with the following differential: eosinophiles 33 per cent, basophiles 2 per cent, stab cells 4 per cent, segmented cells 35 per cent, lymphocytes 25 per cent, and monocytes 1 per cent. On October 8 and November 1, 1943, the eosinophiles fell to 12 per cent and 2 per cent respectively.

Comment. This patient was a known asthmatic with a long history of sensitivity to pollens and dust. An urticarial eruption appeared two days after a normal bronchogram. Eight days following the bronchogram, he developed an acute pulmonary episode characterized by chilly sensations, malaise, substernal pain, and cough. Roentgen examination of the lungs revealed bilateral basal densities more marked on the left side. Marked eosinophilia persisted during the acute illness.

The occurrence of these 3 cases within a relatively short period of time served to center interest upon certain features common to all and differing from other cases of pneumonia. It was noted that in all instances the patients were known asthmatics, that 2 of the cases had a skin eruption resembling hives following bronchography, and that all of the cases developed bilateral basal pneumonia nine, twelve and ten days respectively following that procedure. All of the cases likewise showed increases in total white blood count and polymorphonuclear neutrophiles and 2 of the cases showed striking elevations in the total eosinophile count.

Because of this coincidence of findings, it was considered important to investigate the lipiodol as a possible source of the pneumonia.

Sensitivity tests were done on the 3 patients and a group of 10 control cases. The controls included several asthmatics who had had bronchography with lipiodol without apparent sequelae, as well as other patients on the chronic chest disease wards.

Various methods have been used to determine sensitivity. It was considered important to evaluate these methods of sensitivity testing and consideration was given to work which was done by Pelner[7] and by Archer and Harris.[8] It was determined to use the following tests for sensitivity:

1. Intra-ocular test with lipiodol.
2. Scratch test (forearm) with lipiodol.
3. Scratch test (forearm) with pure poppy seed oil (basic oil of lipiodol).
4. Intracutaneous test with diodrast, an aqueous solution of iodine in organic combination.
5. Intracutaneous test with butyn sulfate 2 per cent, the local anesthetic used in these studies.

Results. The results are summarized in Table 1.

All control cases failed to react. The 3 cases under investigation reacted only to

diodrast injected intracutaneously and Cases I and III reacted to lipiodol by scratch test. Following diodrast, Case I developed a marked erythema of the entire forearm with a large wheal and itching at the site of injection. Case III developed the same local reaction and in addition developed hives of the shoulders and neck. Following scratch tests with lipiodol, both cases developed a local wheal and itching, but the reaction in both cases was not marked. Case II reacted to intracutaneous diodrast with a wheal, but did not react to lipiodol.

TABLE I

RESULTS OF SENSITIVITY TESTS

Sensitivity Tests	Controls (10)	Case I	Case II	Case III
Intra-ocular test: lipiodol	No reaction	No reaction	No reaction	No reaction
Scratch test: lipiodol	No reaction	Moderate reaction: local wheal, erythema, and itching	No reaction	Moderate reaction: local wheal, erythema, and itching
Scratch test: pure poppy seed oil	No reaction	No reaction	No reaction	No reaction
Intracutaneous test: diodrast	No reaction	Marked reaction: local wheal, marked erythema and itching	Moderate reaction: local wheal, and erythema; no itching	Marked reaction: large local wheal, marked erythema and itching; hives of shoulders and neck
Intracutaneous test: butyn sulfate 2%	No reaction	No reaction	No reaction	No reaction

DISCUSSION

The coincidence of the development of bilateral basal pneumonia conforming anatomically to the portions of the lung fields previously instilled with lipiodol, associated with hives and eosinophilia and occurring in known cases of bronchial asthma, seemed to warrant further investigation as to possible sensitivity to lipiodol, perhaps specifically to iodine. Sensitivity tests in the cases reported and in controls seemed to incriminate the lipiodol as the agent responsible for the acute pulmonary episodes. It may be considered that the iodine portion of the lipiodol was the allergen involved, and this seemed to be borne out by the failure of these patients to react to pure poppy seed oil or butyn sulfate, the anesthetic used, and by the marked reaction produced by diodrast, an organic compound of iodine and the less marked reaction produced by the scratch test with lipiodol.

One may conjecture as to what exact pathological process was involved in these cases. Ellis and McKinlay[9] have described what they term "allergic pneumonia," but in the absence of further knowledge of the pathology which these cases represent, it is felt that no attempt should be made to give any descriptive term to this type of pneumonia.

Further, why more than a week passed before the onset of pneumonia in these patients is also a matter of conjecture, although there may be a relationship between the form in which iodine is present in poppy seed oil and its ability to act as a sensitizing agent. It is noteworthy that the interval of time between instillation of lipiodol and

onset of pneumonia in these patients resembled the "delayed reaction" of serum sickness.

CONCLUSION

1. Three cases of bilateral pneumonia following intrabronchial instillation of lipiodol are presented.

2. Evidence is given which would indicate that lipiodol was the cause for the acute onset of pneumonia described.

3. The importance of skin testing individuals, especially those with allergic backgrounds, is emphasized.

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PRIMARY AMYLOIDOSIS OF THE LUNGS

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THE secondary type of amyloidosis, as it occurs in association with some chronic disease, is fairly familiar. In it amyloid is deposited most commonly in parenchymatous organs such as the liver, spleen and kidneys. The primary or atypical form, in which no antecedent condition appears to offer a background for the deposition of amyloid, is far less frequently encountered. It is more prone to affect other organs, the heart and lungs, the skin, the tongue and various mesodermal tissues. The radiological literature is particularly lacking in information on this subject, although a fair number of cases have been reported in other journals, mainly those devoted to pathology.

The characteristics which Lubarsch ascribed to the primary form have been generally accepted as criteria on which a diagnosis of atypical or primary amyloidosis, as opposed to the secondary type, may be based. These have been quoted repeatedly in the literature and are as follows:

1. Almost complete absence of amyloid in organs most involved in typic amyloidosis such as spleen, liver and kidneys.
2. Presence of amyloid in organs and parts not usually involved, such as heart, lungs and skin.
3. The occasional occurrence of tumor-like nodules of amyloidosis.
4. The frequent failure of the deposits to react to the specific stains for amyloidosis.
5. The absence of a preceding or concomitant disease to which the presence of amyloidosis may be ascribed.

Individual cases cannot always be so easily placed in one of these two categories, however. For instance amyloid deposits which occasionally are found in association with multiple myeloma may show a distribution more characteristic of the primary than of the secondary type. To meet such contingencies Reimann, Koucky and Eklund proposed the following classification of amyloidosis:

1. Primary.
2. Secondary.
3. Amyloidosis associated with multiple myeloma.
4. Tumor forming amyloidosis.

They further subdivided primary amyloidosis into a systemic type, a generalized disease of the mesoderm, and a localized variety, in which one or two organs in particular are involved. The case here presented falls into this latter group. Simplicity is perhaps better served by retaining the earlier classification into two forms.

CASE REPORT

The patient, a white male physician, aged sixty, first visited the Department of Radiology of St. Francis Hospital as an out patient in April and May, 1941, with a history of some indefinite abdominal discomfort and diarrhea, and a beginning dyspnea and ease of fatigue. He had had a mild pyelitis in 1934 which had promptly cleared. There were no urinary symptoms at this time but a few red blood cells were found in a centrifuged urine specimen. An excretory pyelogram was done with negative findings. Soon thereafter a roentgen examination of the entire gastrointestinal tract also showed normal findings. Roentgenoscopic and roentgenographic chest examination (Fig. 1) demonstrated a slight enlargement of the left lower part of the cardiac shadow, a slight diffuse aortic enlargement and some accentuation of the bronchovascular shadows in the inferior part of each lung considered to be due to slight peritruncal fibrosis.

In December, 1941 he developed an acute respiratory infection which bore some clinical characteristics of an atypical pneumonia: fever, malaise, cough and pain in the left chest. He recovered sufficiently to resume some work eighteen days later but his convalescence was delayed by weakness, dyspnea and some pre-

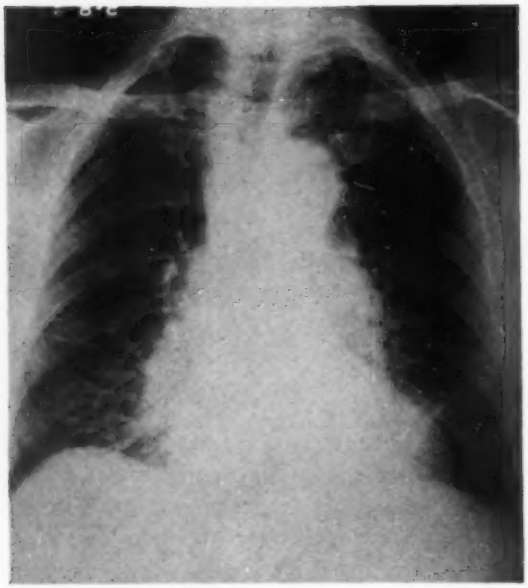

Fig. 1. May 8, 1941. Slight cardiac and aortic enlargement. Moderate enlargement of left and lower right hilar shadows. Early peritruncal fibrosis of lower parts of both lungs. Emphysematous bullae right apex.

mature heart beats. Roentgenograms of the chest on December 20, 1941 (Fig. 2 and 3) showed an increase in the parenchymal shadows plus a left basilar pleuritis. An electrocardiogram showed a lower potential in all leads than previous tracings. Previous electrocardiograms had been made following a transient attack of paroxysmal auricular fibrillation in 1937. An eminent cardiologist was consulted who believed that the condition was probably a mild cardiac weakness due to "progressive narrowing of his coronary arteries" (or occlusion lenta). He advised a few month's rest. This advice was followed and digitalis used with no further clinical improvement.

A roentgenogram on January 9, 1942, showed slight improvement, mainly in the signs of pleuritis (Fig. 4). The patient then spent two months at rest in Tucson, Arizona, where additional studies were made (Fig. 5). These and others of April 17 and May 22, 1942, after his return, revealed an increase in the linear and nodular shadows in both lungs. Thereupon the concept of a pulmonary congestion on a cardiac basis, which had been given some consideration, was abandoned. The roentgen diagnosis was a diffuse pulmonary fibrosis of undetermined etiology and a left pleuritis. Throughout this period dyspnea was the predominant symptom, associated with weakness and left chest pain. The patient denied any exposure to harmful dusts on specific questioning (Fig. 6 and 7).

The possibility of a pulmonary sarcoidosis was considered and when a cervical adenopathy developed a few months later a gland was removed for biopsy. It showed a type of fibrosis considered suggestive but not characteristic

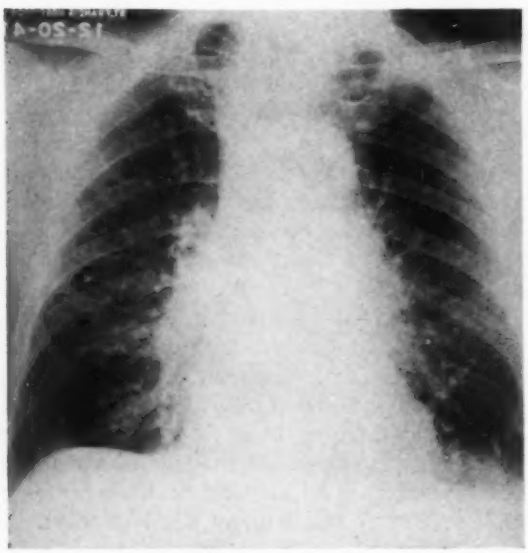

Fig. 2. December 20, 1941. Left basilar pleuritis. Increased diffuse pulmonary and hilar fibrosis.

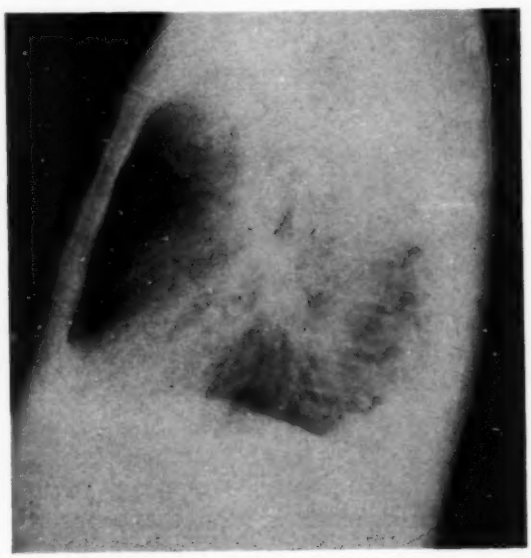

Fig. 3. December 20, 1941. Lateral projection. Obliteration of posterior sulcus.

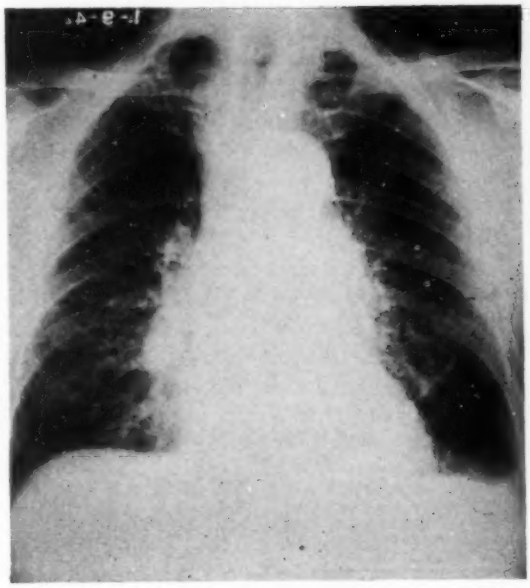

Fig. 4. January 9, 1942. Slight improvement in left pleuritis. Little change in lung parenchyma (heavier exposure).

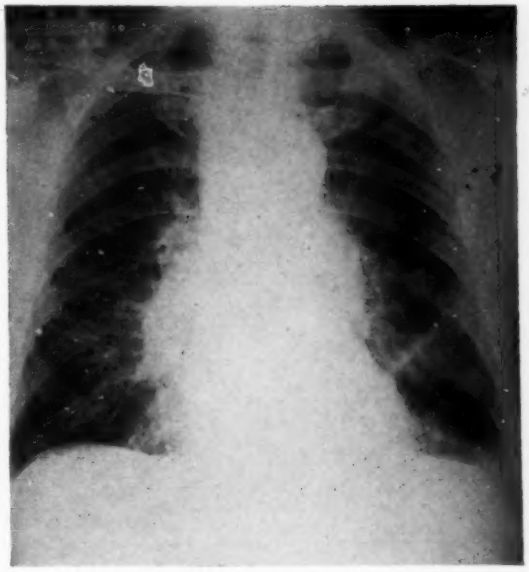

Fig. 5. February 23, 1942. Increased linear and nodular shadows throughout both lungs. Further hilar enlargement. (Courtesy of Tucson Clinic, Tucson, Arizona.)

of that disease. Unfortunately, no specific stain for amyloid was employed. The history and the roentgenographic and pathologic findings were reviewed by several prominent radiologists and internists and, although none ventured a positive diagnosis, the consensus favored Boeck's sarcoid.

Since two radiologists suggested roentgen therapy to the chest on the basis of that tentative diagnosis and because of a reported favorable response of two or three recent cases to that agent (not published), such treatment was

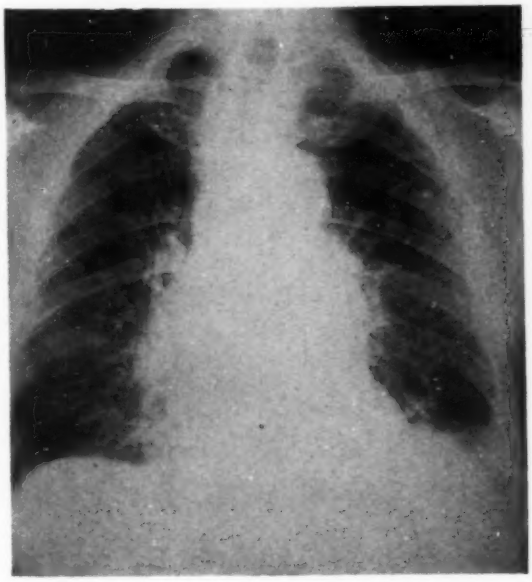

Fig. 6. May 22, 1942. Beginning atelectasis of left lower lobe.

Fig. 7. Lateral projection, same date.

Fig. 8. November 7, 1942. Atelectasis left lower lobe. Slight right, moderate left pleural effusions. Increased hilar, mediastinal and pulmonary fibrosis. Probable pulmonary vascular congestion superimposed.

instituted on November 7, 1942. On that date a roentgenogram showed a further increase in the pulmonary fibrosis, an increase in the left pleuritis along with some atelectasis, and a small bilateral pleural effusion (Fig. 8). Using 200 kilovolts, 10 milliamperes, a 0.5 mm. copper filter, and a target-skin distance of 50 cm., a total of about 1,200 roentgens, measured in air, was delivered to each of four areas, two anterior and two posterior, over the chest. With the same technique 600 r was given to the left chest laterally and 300 r to the lateral part of the right chest. One dose of 200 r was given to each side of the neck. The treatments were usually given at two or three day intervals.

After an apparent slight initial response the patient gradually and progressively became worse. Treatments were discontinued December 1, 1942, and the patient died on January 23, 1943. The last few months were marked by a progressive orthopnea, an unproductive cough and an increasing edema of the lower extremities.

Blood findings were inconclusive. There was a constant leukocytosis of 10,000 to 15,000 cells per cu. mm. from 1934, when it was first discovered, until shortly before the patient's death, when it fell to 6,400. The differential count was usually normal with an occasional lymphocytosis.

Postmortem Examination

Anatomical Diagnoses: (1) primary amyloidosis of lungs, lymph nodes and spleen; (2) hypertrophy of both ventricles and right atrium; (3) generalized atherosclerosis of aorta and peripheral arteries; (4) bilateral hydrothorax and anasarca of both feet and legs; (5) atelectasis of lower lobe of left lung; (6) calcified primary tuberculous complex in lungs and regional lymph nodes; (7) bullous emphysema of right upper lobe of lung; (8) circumscribed left adhesive pleurisy; (9) multiple subserous hemorrhages in the small intestine.

Gross Examination.

The body was that of a moderately well nourished white male of medium size. The skin showed no pathological lesions. There was a definite edema of both feet and the lower halves of the legs. The cervical, axillary and external inguinal lymph nodes were enlarged up to the size of a hazelnut. On opening the abdomen there was no free fluid present. The small intestine was contracted, apparently empty and there were many subserous hemorrhages regularly scattered over the bowel, each being about the size of a tapioca grain.

On opening the chest it became evident that the ribs and the sternum showed a considerable degree of osteoporosis. In both pleural cavities, particularly the left, there was a large amount of clear amber serous fluid amounting to about 3 pints. Both lungs were conspicuous by their dark color, particularly revealed on the surface. The pleura was slightly thickened in some places but not adherent to the chest wall except at the left lower lobe which was fixed to the parietal pleura by one string-like adhesion of the thickness of a pencil. The left lower lobe was completely airless. All lobes of the lungs showed a striking increase in consistency due to the presence of indurated areas, either in the form of small ill defined nodules or strands, black stained by anthracosis. In the apex of the right upper lobe, the lung showed a few thin-walled cavities up to the size of a hazelnut, very closely compacted, as seen in bullous emphysema. The bronchial tree was opened and no tumor was found. The mucosa was reddened and covered with mucus. All bronchomediastinal lymph nodes were enlarged up to the size of a pigeon's egg, black by anthracosis, hard in consistency

with a waxy, dull lustre on the cut surface and imbedded in heavy scar tissue. This scar tissue was amalgamated to the trachea, main bronchi and the large blood vessels. All these structures composed a mass strikingly rubbery in consistency. A calcified area the size of a cherry seed was found in the right lower lobe and calcium deposits in some lower tracheobronchial lymph nodes.

The heart was enlarged, due to hypertrophy of both ventricles. The wall of the right ventricle particularly was much thickened. The mitral valve showed a flat area of thickening particularly in the posterior cusp characterized by a rough, almost verrucous relief and hard consistency.

The liver was normal in size, but the markings indicated passive congestion and probably some fatty infiltration. The gallbladder and bile duct system were negative.

The spleen was large, at least twice normal size, firm in consistency and dark red, showing obscured markings on the cut surface and a dull lustre as seen in diffuse amyloidosis. Scattered through the red pulp were many calcified nodules, round in shape, the largest about the size of a pepperseed.

The adrenals were large, almost double the size of normal adrenals. The cortex was thickened and diffuse yellow.

The kidneys were essentially negative.

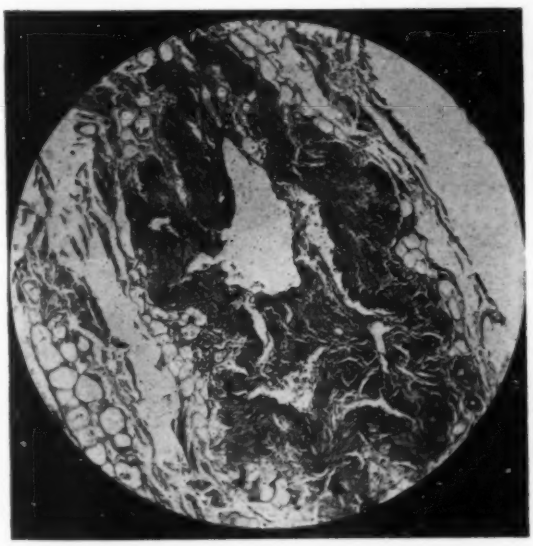

Fig. 9. Photomicrograph showing severe amyloidosis of blood vessel wall and of supporting fibrous and adipose tissue.

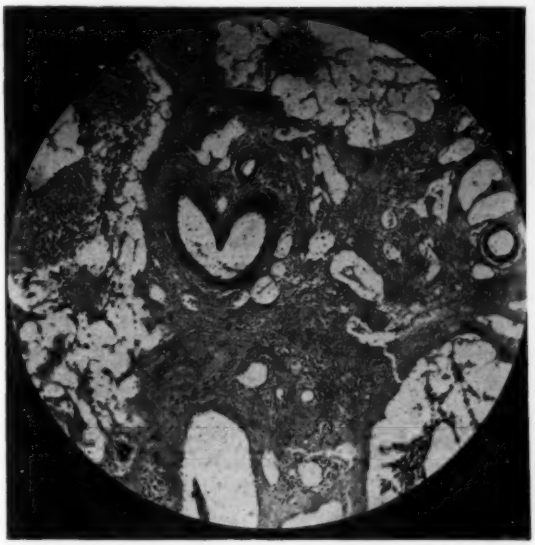

Fig 10. Photomicrograph showing mediastinal lymph node structure largely replaced by fibrous tissue infiltrated by amyloid.

The abdominal and pelvic lymph nodes were all enlarged to varying degrees, the largest attaining the size of a pigeon's egg. They were rubbery in consistency and striking by the dull, waxy lustre of the cut surface. On touching the cut surface slightly one had somewhat the sensation of fine sand. The color of the lymph nodes on cross section was partly pinkish, partly grayish-white. There was no normal marking discernible on the cut surface. The fat tissue surrounding these lymph nodes also was indurated.

The intestinal canal did not show essential changes except the subserous hemorrhages mentioned above.

Histological Examination

Lung: The alveolar walls and lung septa are thickened due to deposits of amyloid. The walls of blood vessels are filled with amyloid. There are many "heart failure" cells and many deposits of anthracotic pigment.

Mediastinal Connective Tissue: Sections show an almost solid infiltration by amyloid (Fig. 9).

Lymph Nodes: The normal structure is almost gone due to large deposits of amyloid (Fig. 10).

Jejunum: There is a small circumscribed deposit of amyloid in the submucosa.

Spleen: There is an almost diffuse deposition of amyloid throughout the red pulp. The follicles are atrophic and apparently without

amyloid. The walls of the blood vessels also contain amyloid.

Heart: In some parts there is much fibrosis which contains amyloid. The muscle fibers are hypertrophic and show fatty degeneration. There is much lipofuscin at either pole of the muscle nuclei. Sections of a thickened valve (mitral) show deposits of amyloid and much fibrous tissue. There are small deposits of amyloid on the inner surface of the pericardial sac.

Kidney: No amyloid.

Liver: No amyloid.

Adrenal: There is much lipoid present in the cortex and it is evenly distributed. There are amyloid deposits in the fibrous tissue surrounding the cortex. There is amyloid in the walls of the central adrenal vein.*

DISCUSSION

Primary amyloidosis is a very uncommon disease. For this reason and because the symptomatology is so varied, depending upon the location of the amyloid deposits, the diagnosis has rarely been made clinically.

The tongue is a frequent site of involvement and here the condition results in an enlargement of the tongue which simulates cancer. Dyspnea is a very prominent feature of pulmonary amyloidosis but of course it is a very common symptom in many diseases. Extensive amyloid deposits in the heart may lead to cardiac failure, which also results in dyspnea, and again amyloid tumors of the larynx or trachea will produce a form of dyspnea. Purpuric types of hemorrhage have been described occurring in the skin and the mucous membranes, as well as hematemesis, melena and hematuria. Such bleeding has been ascribed to vascular infiltrations. However, amyloid deposits in the walls of blood vessels, especially the smaller arteries and veins, is a very common finding in this disease but bleeding is seldom reported.

Gastrointestinal deposits may also result in either constipation or diarrhea, abdominal pain or vomiting. The case history here recorded includes a complaint of abdominal[1] distress and at least one period of mild diarrhea but there was no gastrointestinal amyloid except one small deposit in the jejunum. Primary involvement of bone is rare. One case has been reported in which a pathological fracture of the neck of the femur occurred. Deposits in muscles, tendons and joint capsules have been more frequent and have caused the symptoms of stiffness, painful motion of extremities, back pain and difficulty in walking.

A considerable number of skin lesions have been described. These have been lichenoid lesions and plaque-like hyperkeratoses varying from pink to yellow or light brown and to a normal color. Even where such lesions were biopsied the true diagnosis was not established in more than one instance, to be arrived at during subsequent autopsy. This has been due to atypical staining reactions or, more likely, to failure to employ a specific stain for amyloid.

Peripheral nerve infiltrations have been recorded specifically in 2 cases, resulting in a progressive muscular weakness. Primary systemic amyloid, as the name implies, involves many organs and hence produces a variety of symptoms. Even the localized type, not including a few local amyloid tumors, show slight disseminated amyloid deposits in addition to the primary involvement of one or two organs. It is easily understood, therefore, why the clinical diagnosis of this disease is so rarely made. It is quite possible, however, that with increasing awareness of the possibilities of this condition, and with more widespread use of the biopsy and the Congo red test the condition may be more commonly recognized in the living patient.

Whether roentgenography will contribute materially to our success in evaluating these cases correctly antemortem remains to be demonstrated. So few observations are yet available that it is impossible to establish any positive diagnostic criteria or to do more than relate the few findings of individual cases.

* I wish to express appreciation to the pathologist, Dr. J. E. Kraus, for pathological diagnoses and descriptions and other assistance.

Several descriptions of roentgenographic findings in gastrointestinal amyloidosis are available. Lubarsch* in 1929 mentioned that one case having amyloid deposits in the gastric musculature showed roentgen signs of a pyloric stenosis and was diagnosed a carcinoma of the pylorus. Another case showed only a disturbance in the passage of the opaque meal through the stomach. Clausen* in 1935 described a narrowing of the prepyloric canal 2 cm. long and 1 cm. in diameter. Proximal to this there was increased peristalsis. The pylorus and duodenal bulb were normal and there was no delay in emptying. A diagnosis of an infiltrative process of the gastric wall, probably scarring or tumor, was made. Autopsy revealed extensive amyloid degeneration of the musculature and microscopic deposits in the mucosa. Heeren described a colonic lesion, apparently of the secondary type. This showed irregular jagged contours of the sigmoid, descending and transverse colon and a slight narrowing of the lumen when distended. A postevacuation roentgenogram showed an irregular, widened mucosal pattern. An air injection study revealed some haustral folds and only slightly less than normal flexibility. These characteristics the considered distinguishable from those of an ulcerative colitis, which it resembled. He stated that an increase in the size of the lumen could be expected in a late stage in contrast to the narrowing of colitis.

Few reports of bone and joint lesions have appeared. Lengh described involvement of several joints in a case of generalized amyloidosis which he thought secondary to chronic pyelitis. The carpal bones showed a spotty osteoporosis with smooth contours. There were bean-sized, faintly opaque shadows in the soft tissues of the wrist and knee which proved to be amyloid deposits containing calcification. Several of these were free in the joint space.

Koletsky and Stecher, in reporting a case of the primary systemic type, reproduced a roentgenogram showing a pathological fracture of the femoral neck with considerable fragmentation and absorption of bone. Other roentgenograms of the same case were reported to show a general demineralization of both hands, one knee and one shoulder. There was fragmentation about the greater tuberosity and a slight downward displacement of the head of the humerus. Clinically these joints showed a firm, irregular, non-tender enlargement. At autopsy a very large mass of amyloid was discovered replacing the bone of the neck of the femur and extending into surrounding tissue.

Very few cases of pulmonary involvement of significant degree are reported and in at least 2 such cases no roentgenograms were made. A few instances of microscopic amounts of amyloid in the lungs are reported with negative roentgenographic findings. Nevertheless, there is a consistency in the few positive findings available that may be significant. Sappington, *et al.* have recently reported a case of primary amyloidosis of the lungs with the following roentgenographic findings: " . . . some enlargement of the cardiovascular silhouette, and the lung fields were indefinitely hazy." Michelson and Lynch presented a case of systemic primary amyloidosis with marked involvement of the lungs and mediastinum. A roentgen examination of the chest is reported to have shown a slight bilateral pleural effusion and "a diffuse infiltration throughout both lungs following the bronchovascular trees and suggesting some type of pulmonary congestion of rather extreme degree or an infiltrating process. There was a large mass in the region of the right hilum." This roentgenogram was reproduced and shows considerable similarity to those of our case, as do the respective pathological findings within the chest. In that case a roentgenogram less than two years before was reported negative.

In the case here reported the first roentgenogram, twenty months before death, already showed some bilateral enlargement of the hilar shadows and a linear and finely

* As quoted by Heeren.[14]

nodular peritruncal infiltration widely disseminated in the parenchyma of the lungs. Seven months later these findings were intensified considerably. At that time a left basilar pleuritis developed, but this was thought to have improved slightly two months later (February 23, 1942). One year after the first examination (May 22, 1942) a gradual progression of the hilar and parenchymal fibrosis was shown but by then some atelectasis of the left posterior lung base had developed. This slowly increased during the remaining eight months of life. The last roentgenogram, over two months before death, revealed all of these characteristics much increased in degree and extent, but apparently complicated by beginning heart failure. There was also increased density and loss of sharp outline of the superior mediastinal shadow reflecting the severe fibrosis of the mediastinum within which were discovered many amyloid deposits.

It is possible to reconstruct from these roentgenographic changes, in the light of the autopsy findings, the gradually progressive deposition of fibrous tissue and amyloid material in the lungs, hila and mediastinum over a period of certainly more than twenty months. The fact that bed rest and digitalis had no effect on its course served to distinguish it from a pulmonary congestion and edema from cardiac decompensation. A careful history eliminated the possibility of a pneumoconiosis. The similarity to a pulmonary sarcoidosis was striking. In such cases the use of a biopsy, if material is available, and the Congo red test should permit differentiation when the possibility of a primary amyloidosis is kept in mind, as it was not in this instance. The unremitting retrogressive course has been characteristic of all cases of this disease.

CONCLUSIONS

1. The report of a case of primary amyloidosis involving primarily the lungs, mediastinum, lymph nodes and spleen is added to the slowly growing list of such cases.

2. Roentgenographic characteristics of the intrathoracic form of this condition in this case are described, with a review of roentgen findings previously recorded in these and other organs.

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THE ROENTGEN DEMONSTRATION OF AN ABERRANT PANCREATIC NODULE IN THE STOMACH

REPORT OF THREE CASES

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ANOMALOUS formation of the pancreas, though by no means common, is far from rare. The anomalies may take one or more of the following forms:

1. Separation of the part of the head known as the uncinate process, which then forms a lesser pancreas.

2. A growth of the pancreas around the duodenum which it may encircle for a short part of its course (annular pancreas).

3. Variations at the termination of the main pancreatic duct: (*a*) partial or (*b*) non-joining with the common bile duct.

4. Displacement of the main pancreatic duct termination by a congenital diverticulum of the duodenum near the ampulla of Vater.

5. Accessory or aberrant pancreas. It is this last anomaly to which this report is devoted.

Acessory glands (accessory or aberrant pancreas) appear as rounded, flattened tumors varying in size up to 4 cm. They may be single or multiple. They may occur anywhere in the stomach, small bowel, mesentery, or omentum. They have been found within duodenal or Meckel's diverticula. They may have independent excretory ducts sometimes opening on papillary elevations. They seldom contain islands of Langerhans, but if present they are usually deformed. Small myomas (adenomyomas) may develop around aberrant pancreatic nodules.

Most pathologists and embryologists are of the opinion that these pancreatic nodules may occur anywhere in the stomach, small bowel, mesentery, or omentum, corresponding to the primitive anlage of the pancreas. They develop in the form of single or multiple small cell rests. In 1934 King and MacCallum proposed that adult tissue, namely the intestinal epithelium, could retain its metaplastic powers so prevalent in the embryonic stage. In this way they believed that they could explain the presence of aberrant pancreatic tissue. They offered several plausible, though not conclusive, reasons to substantiate their views. This hypothesis, though apparently little accepted, does offer interesting speculation.

The demonstration of aberrant pancreatic tissue in the gastrointestinal tract has been recorded in the literature for a great number of years, so that at the present time there are over 300 individual cases on record. For the most part these have been discovered as incidental findings at autopsy.

In 1924 Vigi and Gamberini, after studying numerous pathological specimens, predicted that aberrant pancreatic tissue could be demonstrated roentgenologically during life and they went further to describe what they believed would be the characteristic findings. These, however, are not pathognomonic. Since then a definite abnormality has been noted on roentgen examination of the gastrointestinal tract by Branch and Gross, King and MacCallum, Faust and Mudgett, and others. In no case was the true histopathological nature of the lesion diagnosed prior to operation or autopsy. In most instances the roentgen diagnosis was carcinoma.

A careful search of the American and British roentgenological literature discloses that very little has been written on this

Fig. 1. Case 1. Roentgenograms showing the 1.5 cm. spherical defect.

interesting subject. It was therefore believed desirable to record the following cases, especially so because the illustrations depict the anomaly very clearly.

CASE REPORTS

Case 1. The patient was a white male, aged thirty-one, who had no complaints until approximately ten months ago. At that time he began to notice gnawing pain in the epigastrium which usually occurred twenty to thirty minutes after meals. This pain was easily relieved by the ingestion of milk. Recently the pain became sufficiently severe to awaken him at night. There was occasional vomiting but no hematemesis or melena. There was a concomitant weight loss of 14 pounds.

The physical examination was negative except for the abdomen. This was flat and showed no masses. The liver, kidneys and spleen were not palpable. There was tenderness in the midepigastrium with the maximum point 2 to 3 cm. to the left of the midline.

A gastrointestinal roentgen examination revealed a partially obstructing duodenal ulcer. As an incidental finding a spherical polypoidal mass 1.5 cm. in diameter was noted in the pylorus, just proximal to the pyloric sphincter on the anterior wall. The mass was constant in size, its borders were smooth and it rested on a flat sessisle base. The general and local contour and function of the stomach were unaffected.

A few days later a second roentgen examination was performed. The findings were identical with those of the first.

A week later a gastric resection was done removing the distal 60 per cent of the stomach. This included the mass noted in the roentgen examinations.

Pathological Report. Gross description. The specimen consisted of a segment of stomach, previously opened along the lesser curvature. The serosa was smooth, pale and intact. The wall was of normal thickness. On the anterior wall there was a cylindrical tumor mass measuring 1.5 cm. in diameter which projected into the lumen of the stomach. The overlying mucosa was intact. Its center was slightly umbilicated. The mass was freely movable and apparently attached to the mucularis. The gastric mucosa elsewhere was normal. Microscopic description: the mucosa over the tumor mass was thinned but intact and heavily infiltrated with lymphocytes. In the mucosa and submucosa were small collections of glandular structures separated from one another by fibrotic bands of variable thickness. These acini were small, regular, and lined by pyramidal cells. The lumina were often indistinct or completely lacking. Occasionally associated with these acini were small ducts lined by a single layer of columnar cells. No connection between the ducts and acini was discerned. Elsewhere,

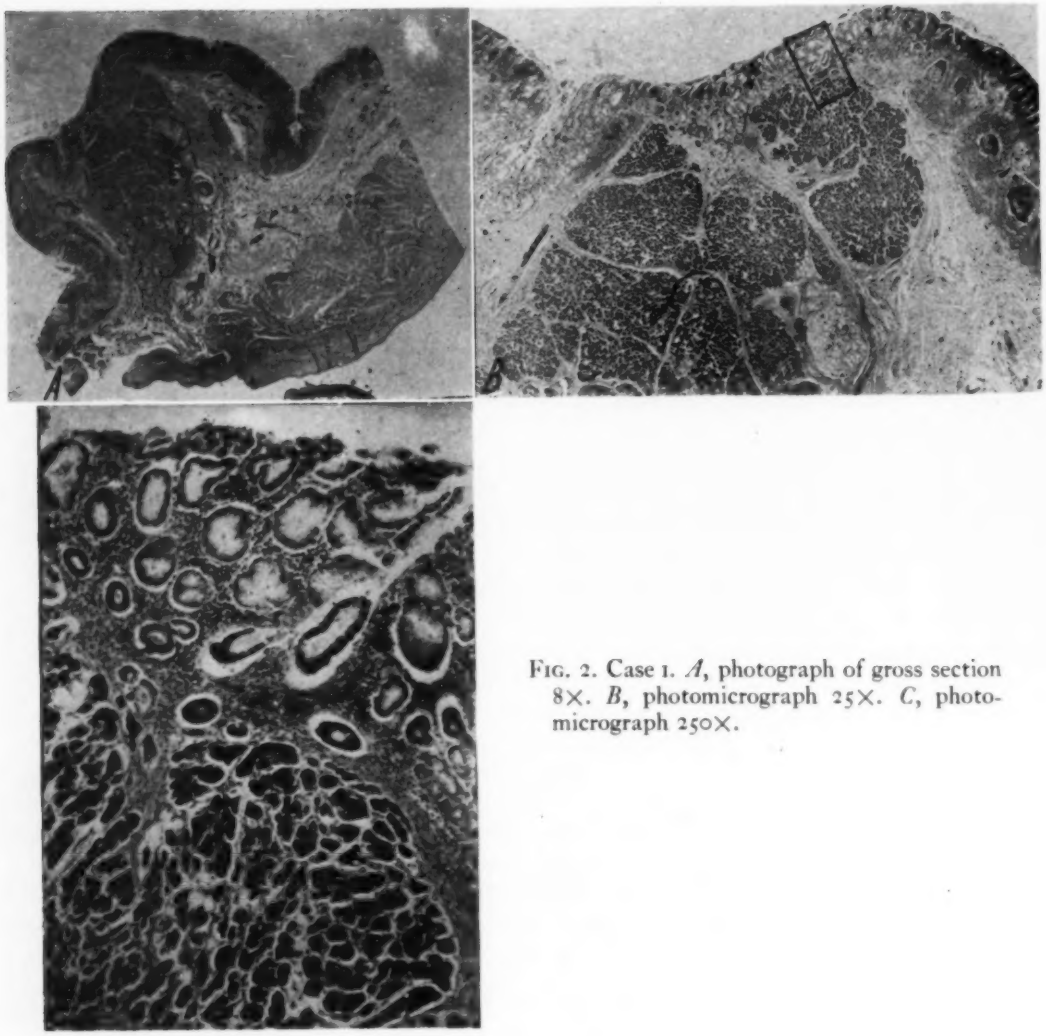

FIG. 2. Case 1. *A*, photograph of gross section 8×. *B*, photomicrograph 25×. *C*, photomicrograph 250×.

there were groups of larger, irregular similar ducts surrounded by clusters of typical mucous glands whose mucin failed to stain by either the Mallory-eosin, methylene blue method or Masson's trichrome stain. An occasional goblet cell was noted in the lining epithelium of some of the ductal structures. Although the glandular foci were confined chiefly to the submucosa, some were found in the mucosa and in the muscularis. The histopathological picture was characteristic of pancreatic tissue. No islands of Langerhans were found. There was no evidence of cellular proliferation. Microscopic diagnosis: Aberrant pancreatic rest of stomach.

CASE II. Patient was a male, aged fifty-five, who had a two year history of epigastric discomfort which was relieved by food. There had been a 20 pound weight loss in one year. Physical examination was negative.

Gastrointestinal roentgen examination revealed a scar-deformed duodenal bulb with no direct evidence of a niche and no obstruction. On the lesser curvature of the pars media of the stomach a sessile mass 1.7 by 1.6 cm. was noted. It was freely movable but no mucosa folds were demonstrated over it. Peristalsis was normal. Roentgenographically the mass appeared benign, but because of the patient's age, history of weight loss and mucosal disturbance it was decided to do an exploratory operation. A resection was done.

Pathological Report. Gross description: On the lesser curvature of the pars media there was

Fig. 3. Case II. Roentgenogram showing the pancreatic nodule.

Fig. 4. Case III. Roentgenogram showing the pancreatic nodule.

found a 1.5 by 1.5 cm. sessile mass with the mucosa over it intact but stretched and thinned. Microscopic description: The mass consisted of a collection of pancreatic glandular tissue extending down into the submucosa. Only a few small ducts were seen. No islands of Langerhans were seen. Microscopic diagnosis: Aberrant pancreatic nodule.

Case III. This patient was a male, aged fifty-one, who had a one year history of gallbladder disease. For the past month he had had frequent attacks of gallbladder colic. Physical examination was negative except for diffuse tenderness in the right upper quadrant.

Roentgen examination revealed a 1 by 1.5 cm. opaque calculus in the gallbladder. In the stomach a soft pliable mass about 2 by 3 cm. was noted in the lower portion of the body on the anterior wall. The rugal folds were intact and thinned, but separated. Peristalsis was normal. At operation the surgeon decided to do a resection.

Pathological Report. Gross description: There was a 3 by 2.5 cm. size mass on the anterior wall of the pars media of the stomach and the mucosa over the mass was intact. Microscopic description: Aberrant pancreatic glandular tissue was found.

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REPORT OF A CASE OF RUPTURED STOMACH IN AN INFANT THREE DAYS OLD*

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A RUPTURED viscus in an infant is a condition which is seldom encountered and from a review of the literature it is evident that rupture of the stomach in young infants is quite a rare condition. Also, in each case reported, a rupture of the stomach has been considered secondary to the presence of gastric ulcer. Postmortem statistics show that ulcers in children are absolutely and relatively of uncommon occurrence as compared with the incidence in adults. The cases are seldom recognized clinically early and usually are diagnosed only after perforation of the stomach or peritonitis has developed.

Theile in 1919 made a comprehensive review of the subject of ulcers of the stomach of children. He found reports of 248 cases of ulcers in childhood with very few in the neonatal period. He discovered 185 cases of ulcer of the duodenum in children were in boys and 98 were in girls. Sixty-one of the ulcers in his series were not located definitely or differentiated as to in which sex. Bowes was of the opinion that gastric ulcer in infancy predominated in the female.

Perforation of the stomach occurred in only 5 of Theile's cases of ulceration of the stomach and the age of onset of symptoms varied between twelve hours and eleven days in those cases in the neonatal period. The ulcers were described in some cases as having round and sharp cut edges and in others as having torn edges. They were single in all of the cases reported except two. In those cases in which histopathological examinations were made of the stomach at the site of perforation, the changes described were those of necrosis and hemorrhage into the tissue near the point of rupture. A cellular reaction similar to that found in peptic ulcer was not found nor were the evidences of embolism or thrombosis found.

Hibbard reported an interesting case in a three months old female infant with a diphtheritic membrane in the throat. The infant had a perforating gastric ulcer with hemorrhage. Histopathologically the ulcer appeared to be an acute process due to disturbance of circulation of the stomach produced by hyaline thrombi with resultant necrotic mucous membrane which was digested away by gastric juice. There was no granulation tissue or chronic inflammation, and he believed that the ulcer may have started as a diphtheritic lesion of the gastric mucous membrane.

The etiology of such ulcers is quite obscure. Adler believed that it was difficult to explain the occurrence of ulcers in the first few days of life on any other basis than that during parturition a hemorrhage into the mucosa occurred with subsequent autodigestion. An infectious origin in children seems quite possible, however. One author regarded the mechanism of their formation as a local disturbance in circulation which, from the action of the gastric juice, led to necrosis and ulceration. He mentioned as causes of this congestion, embolism, thrombosis, vascular disease, direct injury to the mucous membrane and nervous influences. Also, focal lesions of the hypothalamus and congenital defects in the structure of the stomach have been implicated.

Theile believed that in the adult one sees a primary type of ulcer, whereas in the child a secondary type of ulcer is found. The causes of the latter, Theile lists as melena neonatorum, marasmus (usually causing duodenal ulceration), infectious ulcers such as are seen in conjunction with syphilis or tuberculosis, burns, nephritis and uremia, congestive and catarrhal

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processes in the mucous membrane (follicular gastritis), or direct trauma. Of Theile's 248 cases, 64 were due to melena neonatorum, 60 to marasmus, 31 to burns, 21 to tuberculosis, 26 to other infections, and 2 to uremia.

Fenwick recognized five varieties of gastric ulcer: (1) hemorrhagic erosions as a result of congestion of vessels of the mucosa due to cardiac valvular disease, dilated heart, pulmonary disease, rarely cirrhosis of the liver, scurvy, and blood dyscrasias; (2) follicular gastritis, due to inflammation of solitary lymph glands with subsequent necrosis and ulceration; (3) tuberculous ulcers; (4) malignant ulcers; (5) true peptic ulcer, either acute or chronic and varying grossly from the adult type in not always being round and punched out.

Direct trauma to the mucous membrane of the stomach may be considered as a possible cause in some cases because a tube might be used for gavage or lavage. The use of a tube was noted in some reported cases before any abdominal distention appeared. However, it seems highly improbable that a normal mucous membrane could be damaged by a soft rubber catheter. Similarly, in another case, a tube was introduced into the stomach and the trauma in this case was thought *not* to have caused rupture of the stomach because signs of peritonitis developed before the tube was introduced.

CASE REPORT

A case of perforation of the stomach in an infant was observed in the Hospital of the University of Pennsylvania in August, 1941. The condition occurred in a three day old female infant seen in the hospital nursery following a precipitous delivery. The mother, a colored female, aged twenty-three, was a multipara with two previous normal full term deliveries, the last child having been delivered two years previously. Both of the children were living and well.

The mother's prenatal course was normal except for some pretibial edema in the latter months of her pregnancy. Her blood Wassermann reaction was negative as was a smear for gonococci. The present pregnancy was at term, presenting left occiput transverse; and the following delivery was normal in all respects on August 21, 1941.

However, the infant was cyanotic at birth and *received intratracheal oxygen* for approximately five minutes. The child had attained forty-one weeks of maturity, there were no apparent malformations, and only a slight moulding of the head. The weight of the baby was 3,300 grams and the length 51 centimeters. The infant nursed quite well going to the breasts on August 22, 1941, and seemed to be perfectly normal postnatally. On the morning of the third day following delivery, however, the child was found to have a moderate abdominal distention and its temperature rose to 100° F. Peristalsis was present and active and meconium was passed by rectum. Nevertheless, the distention increased during the day and it appeared to embarrass respiration.

Roentgenoscopic and roentgenographic examinations in the horizontal and erect postures at that time revealed what was thought to be a perforated viscus since there was subdiaphragmatic air with a fluid level in the abdomen (Fig. 1 and 2). It should be recorded that in the horizontal posture the true nature of the abdominal distention could not be ascertained.

An emergency laparotomy was performed with a preoperative diagnosis of perforated viscus. A right rectus incision was made and a large amount of gas and bloody fluid were evacuated from the peritoneal cavity. There was no clue as to the location of the perforation of the hollow viscus so the intestinal tract, which came into view immediately, was examined first. There was no evidence of perforation of the small intestine but it was possible to find material coming down from the upper abdomen, an opening was found in the anterior surface of the stomach near the lesser curvature. This opening was about 1 cm. in length. There was very little induration about it and there appeared to be no obvious explanation for its presence. The gastric contents were aspirated from this neighborhood and the opening in the stomach was closed with two rows of continuous catgut sutures of No. 5 chromic catgut. The peritoneal cavity appeared then to be as clear in this area as in any other area, and it was decided not to drain. The small intestine was replaced in the peritoneal cavity and the wound was closed with steel wire in one layer.

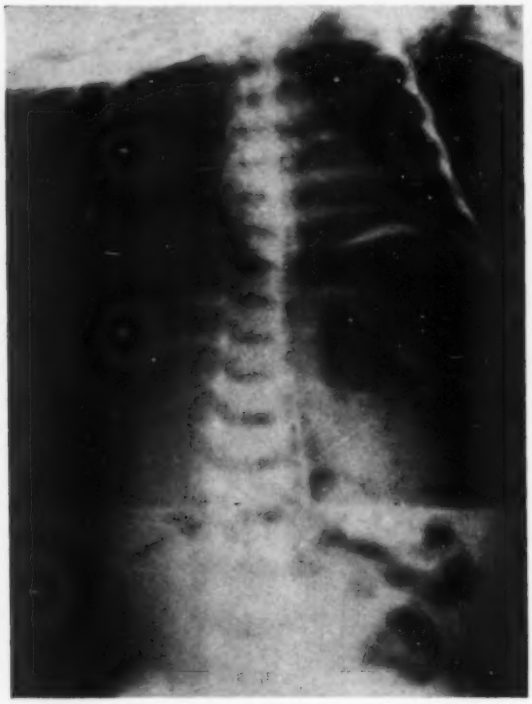

FIG. 1. Pneumoperitoneum with fluid level due to a ruptured viscus.

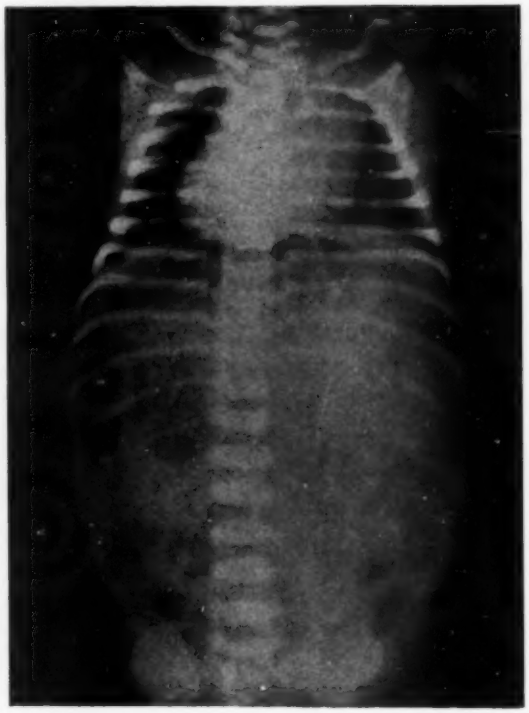

FIG. 2. Same patient as Figure 1. The examination was done in the horizontal posture. One does not obtain any idea of the real cause of the distention. The illustrations (Fig. 1 and 2) indicate the importance of the use of the horizontal beam in recording roentgen manifestations in distention of the abdomen.

The patient returned to the ward in fair condition.

The postoperative course of the infant was gradually retrogressive. The child took no nourishment by mouth and its weight decreased to 3,100 grams. Two days later the infant died.

At autopsy, the most significant findings were in the gastrointestinal tract. The intestines were adherent to each other, to the peritoneal surfaces and to other organs. They were covered with a fresh brownish exudate, most marked in the right upper quadrant. The small bowel was markedly distended with gas.

The stomach showed an intact suture line on the lesser curvature. On the greater curvature, plastered against the spleen, was a defect in the gastric wall about 0.5 by 0.5 cm. There was a small amount of submucosal hemorrhage about the perforation so that it appeared to have had an antemortem occurrence. The remainder of the gastrointestinal tract was normal except for two raised white areas on the serosal surface of the first portion of the ileum.

Microscopic section of the stomach through the area of the operative incision showed the wall to be pulled together by sutures, but there was no real union of the edges, which were covered with necrotic exudate containing bacterial colonies. The submucosa was edematous and congested, but no intrinsic disease was apparent. The serosa was covered with a fibropurulent exudate. The section from the edge of the posterior perforation showed no further lesion.

Therefore, in summary, this infant had two perforations of the stomach soon after birth and died as a result of a fibropurulent peritonitis on the fifth postnatal day. One perforation was repaired surgically but another on the posterior well of the stomach was not found until autopsy. The appearance at autopsy suggested that this latter perforation was present at the time of operation and was overlooked due to its positon and the fact that when one perforation was found and closed, an in-

adequate search was made for another perforation. No definite etiological factor was demonstrated, and there seemed to be no intrinsic disease of the stomach. Perhaps a possibility is that ruptures were caused by the pressure of the oxygen used intratracheally at the time of delivery. Nevertheless, even if the soft rubber catheter which was used for the intratracheal introduction of oxygen (and which seemed actually to have been in the trachea because of the response of the baby) had passed into the stomach, it seems somewhat improbable that the normal mucosa would be perforated in two places. In the light of the well known occurrence of so-called spontaneous rupture of the intestine in the newborn infant, which is probably due to obstruction and trauma during birth a similar mechanism perhaps should be considered here. No such case has been reported, however.

Nevertheless, the significant fact remains that the diagnosis of a ruptured viscus should not be overlooked in an infant merely because of the patient's age. Likewise, in making a roentgen examination of an infant, one should not rely entirely on an examination in the horizontal posture if a vertical beam of roentgen rays is being employed. In this instance, such an examination, which is conventional, would not have revealed the true nature of the condition (Fig. 2). It is recommended, therefore, that at least one exposure be made in the erect posture (Fig. 1).

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Fig. 3. Lateral view showing the free air in the peritoneal cavity and the distention of the small intestine.

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ROENTGEN DIAGNOSIS OF RETROPERITONEAL LIPOMA*

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LIPOMATOUS tumors are characteristic though not frequent growths of the retroperitoneal space. They may occur in early childhood but usually appear in the fifth to sixth decade and are more frequent in women than in men. They may originate on either side of the vertebral column, but more often above the level of the kidney. They grow very slowly and sometimes reach a huge size (63 pounds, Boyd), the yellow, soft, fluctuant mass of fat thus filling the abdominal cavity from pelvis to diaphragm. Smaller ones tend to be spherical, sometimes lobulated. Often there are fibrous septa. The surface of the tumor is covered by the peritoneum of the posterior abdominal wall. Usually there are no peritoneal adhesions.

Microscopic examination reveals the characteristic large cells of neoplastic fat tissue. Myxomatous and fibrous areas are not infrequent, and about one-third of recorded cases display sarcomatous structures. Pemberton and Whitlock observed recurrent growth in approximately one-fourth of operated cases. The presence of myomatous and cartilaginous areas and the tendency toward recurrence, whether the microscopical structure is benign or malignant, influenced some investigators to place these tumors closer to teratomas than to lipomas.

Clinical symptoms are indefinite. The slow growth of the tumor and the adaptability of the soft, fatty tissue to the contour of surrounding structures provoke no symptoms in the early stages. If the tumor achieves vast proportions, its weight may cause abdominal distress, a sense of fullness, and occasionally constipation and backache. A large abdominal mass, of course, is constantly palpable. There are no laboratory tests to suggest the presence or absence of lipoma.

According to available information, accurate roentgenological diagnosis of retroperitoneal lipoma is unreported. These neoplasms have often been mistaken for kidney tumors or, if soft enough to fluctuate, for ovarian cysts. It seems to me, however, that several peculiarities should make the diagnosis possible. The following characteristics are specific to the extent that they may be regarded as basic considerations for diagnostic studies:

1. Displacement of the kidney without gross impairment of its shape and function.
2. Displacement of the abdominal viscera, but no evidence of organic lesions of the intestinal wall.
3. Expansive, not infiltrative, tumor growth.
4. Palpable, soft, sometimes fluctuating tumor. As a rule, no local tenderness.
5. Negative digital pelvic findings.
6. In contrast to the size of the tumor, mostly no signs of malignancy.
7. In contrast to the marked displacement of parts of the intestinal tract, no evidence of soft tissue shadow of the huge tumor, which could explain such displacement.
8. Occasional increased radiolucency of areas previously occupied by intestinal loops.

Due to the low atomic number of the constituents of fat, its absorption coefficient is lower than that of the surrounding structures. If we consider this value of water as 1.00, the corresponding value of fat is experimentally estimated to be approximately 0.486. The larger the tumor, the greater the contrast. Areas occupied by the tumor may even appear translucent. Dis-

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placement of intestinal loops accentuates the effect. Even in cases where the tumor contains fibrous, myxomatous, or myomatous tissue in addition to fat and there is no real radiolucent area, one still remarks the absence of such a soft tissue shadow as would be expected from the large palpable tumor, were it a cyst, hypernephroma, gravid uterus, or the like.

Laurell expressed the belief that fat-containing tumors of the abdominal cavity might be demonstrated. In one of his cases (a large retroperitoneal liposarcoma), however, the roentgen shadow of the tumor, instead of being less dense, was denser than the shadow of the adjacent structures, indicating that the fat content of this particular tumor was exceptionally low. (See also cases of Regan, Sanes and MacCallum.) Approximately 46 per cent of retroperitoneal lipomas are formed by pure fat tissue. Fifty-four per cent are myxolipomas and fibrolipomas. Thus, about one-half the total number possess the physical characteristics necessary to produce specific roentgen signs.

However, the only roentgenologic descriptions I have found of fat-containing tumors concerned dermoids. Such a case is illustrated in Figure 1.

Fig. 1 (2383—1945). R. R., female, aged twenty-six, with large, round, palpable tumor in lower mid-abdomen. Roentgenograms reveal semicircular shadow of fibrous capsule of tumor. Tumor does not cast a shadow, but is somewhat translucent. Arrows indicate fibrous capsule and teeth. Roentgen diagnosis of dermoid cyst was proved at operation. Removed cyst contained large amount of fatty material, hair, teeth, bone, and cartilage.

CASE REPORTS

Case 1 (A88453). A housewife, aged fifty, was admitted on April 5, 1939, complaining of being "chronically nervous." She had suffered from epigastric distress for the past fifteen years, loss of weight, and constipation. Physical examination revealed abdominal hyperesthesia with tenderness at the epigastrium.

At that time, roentgenograms of the gallbladder and upper gastrointestinal tract showed no evidence of retroperitoneal growth.

A new admission three years later (1942) disclosed a history of abdominal enlargement for one year, as well as additional loss of weight. Physical examination revealed a soft, slightly tender abdomen. A large, irregular, movable mass was palpated in the right mid-abdomen, extending from liver to pelvis. The outlines of the tumor were blunt and soft. Roentgenograms taken at that time are seen in Figures 2 and 3.

On June 16, 1942, Dr. L. R. Chandler removed a large retroperitoneal fatty tumor extending from the under surface of the liver to the pelvis.

Pathological Report (Dr. D. A. Wood). The gross specimen consisted of a lipomatous tumor, ovoid and lobulated, measuring 30 by 25 by 8 cm. and two smaller similar yellow ovoids weighing 160 and 8 gm. Their surfaces were covered by a thin, transparent, smooth membrane. Total weight of the specimens was 2,800 gm.

Histopathological Examination. Part of the tumor consisted of adult, adipose connective tissue cells with only occasional intervening capillaries. In some areas, small, fetal fatty cells with foamy to granular cytoplasm were present.

Diagnosis. Retroperitoneal lipoma.

Patient made a rapid recovery and had no complaints until her third admission on February 24, 1944. At this time, a soft, cystic, palpable mass 6 inches in diameter was again

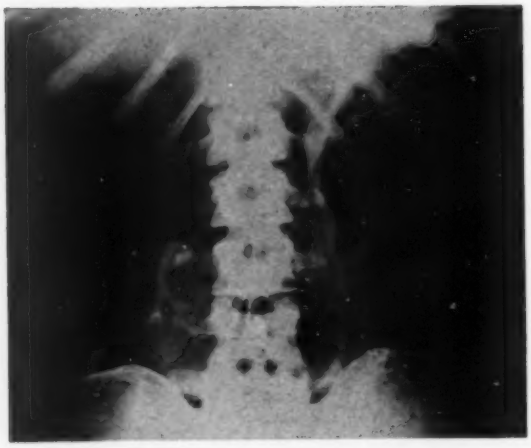

Fig. 2. Case 1. Excretory urogram (1942). Right kidney displaced downward to iliac crest and rotated to right. Dye excretion normal on both sides. Note large translucent area between liver, lumbar spine displaced kidney.

Fig. 3. Case 1. Excretory urogram (1942). Translucency involves upper portion of right psoas shadow and transverse processes of the second and third lumbar vertebrae. Large translucent area corresponds to palpable mass in right abdomen.

felt above and about the area of the right kidney. It extended from the costal margin to the umbilicus and to mid-abdomen. There had been right flank pain for five months and nocturia for the past two years. The diaphragm was elevated on the right. The abdomen was rounded and tender along the right side. The red blood cell count was 4,140,000; hemoglobin 76 per cent (Sahli). Figure 4 shows the roentgen findings.

Fig. 4. Case 1. Excretory urogram (1944). Right kidney displaced downward somewhat more than on first examination. No other changes. Large, translucent area above kidney remains unaltered. Note caudad displacement of gas-distended hepatic flexure, similar to that seen previously.

Exploration revealed most of the recurrent growth located retroperitoneally near the midline and in the transverse mesocolon and gastrocolic omentum, and between the hepatic flexure and the pancreas. There were at least twelve tumors varying from 1 cm. to 6 cm. in diameter. It was deemed impossible to remove all of them; however, one large tumor mass was extirpated by incising the peritoneum at the base of the transverse mesocolon.

Pathological Report (Dr. D. H. Carnes). The gross specimen consisted of a flattened, ovoid, fatty mass 8 cm. in greatest diameter, weighing 120 gm. The mass floated in formalin and ap-

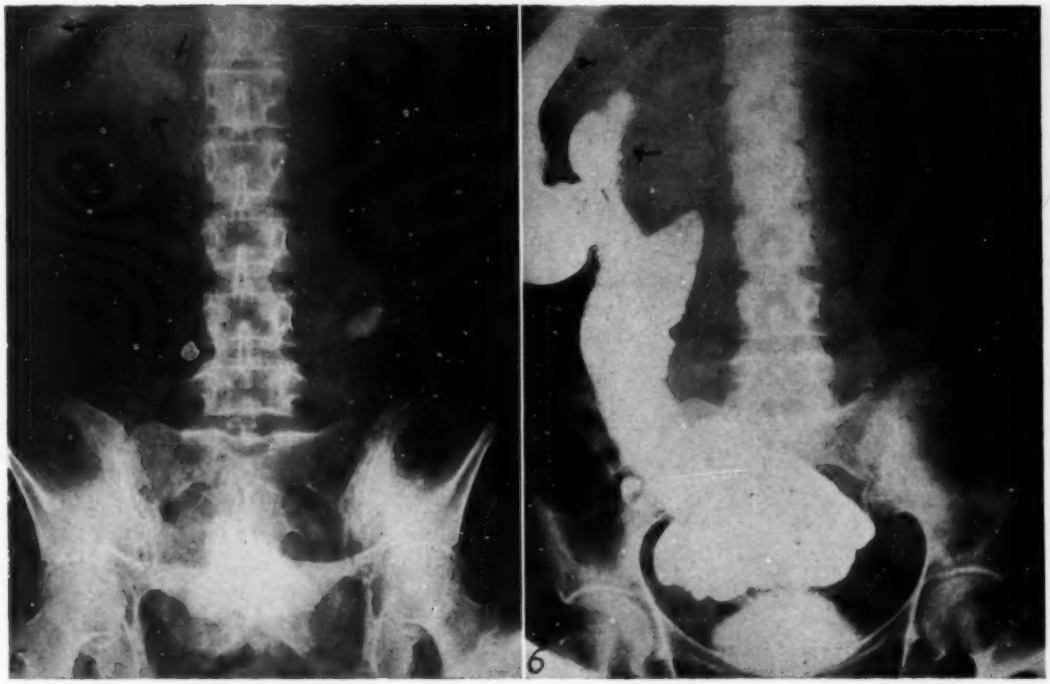

Fig. 5 and 6. Case II. Note remarkable translucency of almost entire left and of middle right abdominal fields. Right kidney displaced craniad, left caudad. It is rotated outward (arrows, Fig. 5). Function good in both kidneys. Upper calyx of left kidney somewhat elongated. Entire intestinal canal displaced toward right. Stomach and duodenum in right upper abdominal quadrant (arrows, Fig. 6). Colon pushed toward right. Gallbladder, containing numerous stones, pushed upward (arrow, Fig. 5).

peared to be composed of uniformly lobulated fat tissue.

Histopathological Examination. The tissue was composed of large, fully developed fat cells forming ordinary looking adipose tissue.

Diagnosis. Lipoma.

Case II (No. 32507/41). A white woman, aged forty-two, complained of distention and a sensation of abdominal fullness and heaviness of eight to ten months' duration. Intensity of symptoms and size of the abdomen have increased during the past months. Previous health was excellent. There was no weight loss.

Physical examination showed a well developed, well nourished woman in apparently robust health. In the recumbent position, the abdomen was moderately distended above the level of the thorax and slightly asymmetrical, the left side being fuller than the right. Abdominal resistance was increased, and a soft mass with indefinite outlines was palpated to the left of the navel. No tenderness was elicited, but the patient complained of some vague distress on deep palpation. Peristaltic sounds were absent except in the right upper abdominal quadrant. Pelvic examination was negative. Roentgen findings were summarized as follows:

Large, abdominal tumor displacing the gastrointestinal tract toward the right side of the abdomen and the left kidney downward and outward. Remarkable translucency of the areas occupied by the tumor indicates lipomatous structure. Displacement of the left kidney points to retroperitoneal origin. Lack of organic involvement of the wall of the gastrointestinal tract is significant of the expansive and noninfiltrative character of the growth (Fig. 5 and 6).

Roentgen Diagnosis. Large retroperitoneal lipoma. Cholelithiasis.

Five days after roentgen examination, the tumor and gallbladder were successfully removed. Laparotomy revealed a very unusual condition. The entire abdomen was filled by an enormous, yellowish-orange, smooth, spherical mass having the appearance of fat tissue. The outer surface was smooth and no adhesions were

Fig. 7. Case II. Gross specimen consisting of spherical, soft, yellowish-orange tumor measuring 20 to 28 by 16 to 18 cm. and weighing 12.5 kg. External surface smooth and shiny, consistency soft, similar to that of fat. Cut sections showed uniform, yellowish fat lobules with few fibrous septa and blood vessels.

present except around the left kidney. The tumor was connected with the left retroperitoneal space, involving the area from the spleen downward to the region of the iliac crest. Operative removal presented no great technical difficulties and the patient recovered uneventfully. Figure 7 is a photograph of the tumor.

Microscopically, the tumor consisted of uniform, large fat cells with a small amount of connective tissue and few blood vessels of various sizes between the lobuli. No structures of malignant growth were evident.

Pathological Diagnosis. Large retroperitoneal lipoma, benign.

Case III (San Francisco Hospital, 6-14-45, No. 98833). Roentgenograms of this case were submitted to me for consultation. Clinical data of this patient, male, aged fifty-one, were not available. Roentgen examination revealed a marked upward displacement of the stomach and first portion of the duodenum. The second and third portions of the duodenum were compressed and horseshoe-shaped. The small intestinal loops were displaced toward the left (Fig. 8). The entire colon, including rectum and sigmoid, was pushed toward the right (Fig. 9). No signs of circumscribed infiltrative lesions were evident in the gastrointestinal tract. The left kidney was displaced upward, and the middle portion of the right ureter was deviated toward the right and anteriorly. The urinary bladder was compressed on the right side. Function was good in both kidneys. Both psoas

Fig. 8. Case III.

Fig. 9. Case III.

and kidney shadows were distinctly visualized (Fig. 10).

Displacement of the kidney, ureter, and duodenum indicated the retroperitoneal origin of the tumor. Displacement of the stomach and colon, and compression of the urinary bladder demonstrated the extraordinary dimensions of the tumor which occupied the retroperitoneal space bilaterally from the diaphragm to the floor of the pelvis. Despite the enormous dimensions of the tumor, no soft tissue shadow was present, indicating considerable fat content of the tumor. The fact that radiolucency was not marked despite the size of the growth suggested that the tumor might contain some collagenous tissue in addition to fat.

Roentgen Diagnosis. Large retroperitoneal lipomatous tumor.

At operation, a tremendous retroperitoneal fibro-myo-liposarcoma was removed.

Microscopical examination of the specimen revealed that most of the tumor consisted of pure fat tissue. The fat cells were large, as in lipoma (Fig. 11). The intercellular septa were very delicate, but blood vessels appeared more numerous than usual. In other parts of the tumor, fibrous and myxomatous structures were present containing cells with huge, dark, irregularly outlined nuclei (Fig. 12). The protoplasm of these cells was translucent, revealing faint eosinophilic precipitates and large vacuoli, the contents of which were not apparent on microscopical examination. These probably consisted of fat. In these parts of the tumor, the intercellular spaces were filled with faintly eosinophilic myxomatous material and delicate small bundles of connective fibers. Structures resembling smooth muscle were also observed.

FIG. 10. Case III.

Pathological Diagnosis (Dr. J. L. Carr). Large retroperitoneal fibro-myo-liposarcoma.

SUMMARY AND CONCLUSIONS

Roentgen signs of three verified cases of large retroperitoneal lipomatous tumors are

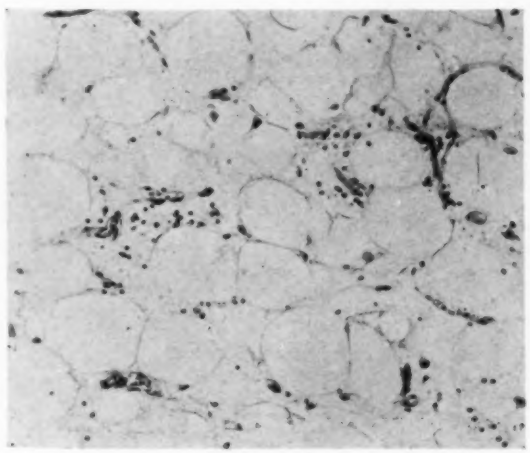

FIG. 11. Case III. Photomicrograph (×300) of lipomatous area of tumor. Hematoxylin-eosin.

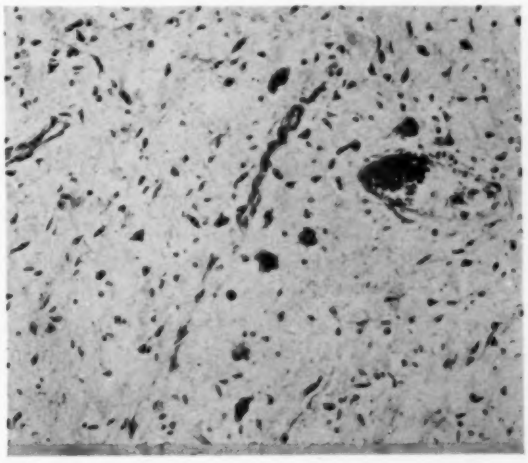

FIG. 12. Case III. Photomicrograph (×300) of sarcomatous area of tumor. Hematoxylin-eosin.

described. In two, accurate preoperative diagnoses were made.

The roentgen appearance was characterized in all cases by radiolucency of the areas involved and by displacement of retroperitoneal and intra-abdominal structures. Maximum radiolucency in two cases was localized craniad and in one caudad to the displaced but functioning kidney. No signs of circumscribed organic lesions of the gastrointestinal tract were present. In the third case, a fibro-myo-liposarcoma, translucency was less marked. A soft tissue shadow of the tumor was absent despite its huge dimensions, reaching from the diaphragm to the floor of the pelvis and displacing the entire gastrointestinal tract.

Approximately 50 per cent of retroperitoneal lipomas are composed of pure fat tissue. Roentgenographic demonstration of them is due to the greater radiolucency of fat and displacement of retroperitoneal and intra-abdominal structures. The translucency above or about the displaced kidney is most easily recognized after the tumor has reached considerable size. At this period of development, the effects of shadow abstraction of the fat (and of displaced and therefore absent intestinal loops) are greater than the effects of shadow summations in the remainder of the abdomen.

The remaining 50 per cent of retroperitoneal lipomas contain varying amounts of myxomatous and fibrous tissue, by reason of which no marked radiolucency is present in areas occupied by such tumors. However, if they attain large size, displacing surrounding abdominal structures, and do not cast adequate soft tissue shadows, it is reasonable to believe they are of lipomatous origin.

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Vol. 56, No. 5

ANKYLOSING SPONDYLITIS

REPORT OF OCCURRENCE IN TWO BROTHERS*

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THE cause of ankylosing spondylitis is not known. Heredity has not been one of the factors seriously considered either for this disease or rheumatoid arthritis. It seems desirable, therefore, to record the occurrence of ankylosing spondylitis in two brothers and to review briefly the literature on the subject.

Ankylosing spondylitis is a particular form of rheumatoid arthritis. The age of onset and the debilitating effects upon the general health are the same for both diseases. The pathological changes in the sacroiliac joints and the apophyseal joints of the spine as described by Güntz[3] are identical with those which are seen in rheumatoid arthritis. They consist of proliferation of connective tissue over the joint cartilage and in the subchondral spaces leading first to obliteration of the joint space and then gradually to complete absorption of the joint cartilage. There is first fibrous union between the two moving elements of the joint which eventually leads to complete bony ankylosis and such rearrangement of bone trabeculae that not the slightest evidence of the former presence of a joint remains. The fundamental lesion is the ankylosis but this process is accompanied and followed by calcification of the intervertebral and intraspinous ligaments until the spine is transformed into a solid immovable bony column. When the dorsal spine is involved, the ribs become ankylosed to the vertebral bodies with resulting fixation of the bony thorax.

The sex incidence is at marked variance from that of rheumatoid arthritis. Ankylosing spondylitis is predominantly a disease of men, less than 10 per cent of the cases being found in women. The onset occurs most frequently in young adulthood. Ehrlich[2] in a survey of 753 cases, noted age of onset from sixteen to fifty but the median age was thirty-one. The disease begins as backache, lumbago or sciatica. The onset may follow an injury or a sprain, and the symptoms are intermittent at first but soon they become constant. Soreness and stiffness of the back continue and progress until the back becomes completely and permanently stiff in one to several years. During the active phase of the disease the patient suffers constitutional ill health with loss of weight, fatigability, irritability and general loss of efficiency. Finally a quiescent stage is reached when the general health returns to normal and the patient becomes as well as he was before his illness except for the mechanical disadvantages of a completely stiff back.

Physical examination shows evidence of loss of weight, stiffness of the spine, flattening of the normal lumbar lordosis, loss of chest expansion, mild kyphosis and a characteristic waddling gait. Not infrequently there is an associated arthritis of the hips with limitation of motion or even complete fixation of these joints. During the active phase of the disease there is anemia and a rapid sedimentation rate.

Early in the disease, roentgenological changes are slight and are limited to osteitis of the sacroiliac joints but these finally become completely fused. Oppenheimer[7] believes the earliest changes are manifest in the apophyseal joints. With special technique employing oblique projections, haziness, narrowing, and finally complete fusion can be observed. Late in the disease, after permanent stiffness of the spine is apparent, calcification occurs in the anterior, lateral and posterior ligaments of the vertebrae as well as the interspinous

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FIG. 1. Photographs of two brothers with ankylosing spondylitis demonstrating posture with moderate dorsal kyphosis, forward inclination of the cervical spine and hyperextension of the head. Case I on the left, Case II on the right.

ligaments producing the typical bamboo spine. The intervertebral discs maintain their normal size and shape throughout the disease.

CASE HISTORIES

The family history of our patients is as follows: the father died at the age of fifty-five of pneumonia, and the mother died at eighty of old age. Of the 8 children, 3 died in infancy and the oldest brother died at fifty-five of unknown cause. Two sisters, aged fifty-four and forty-four are living and well. There was no history of arthritis, rheumatism or backache in the family except as described below.

The father was very deaf, and the mother had normal hearing. The oldest brother, both patients and the two sisters, all of the children now surviving, are hard of hearing. Paul wears a hearing aid.

The brothers, subject of this report, were forced to quit school and go to work at an early age and have done hard work all their lives. They are intelligent, alert and cooperative.

CASE I. Paul is now forty-nine years of age. His arthritis began without known predisposing events at the age of twenty-three years as pain and soreness of the right hip. After a period of several months the difficulty spread to the left hip. Pain was sufficient to produce limping, but it did not keep him from work. After two years, the pain spread to the lower back. He gradually became stooped with pain and suffered from cramps in his ribs. A full back cast was applied for six weeks. Three months after the first cast was removed, a second one was applied for another period of six weeks. The constant stabbing pain in the lower back which he had had for several years disappeared after treatment with casts. In 1925, about seven years after onset, the back was straightened by manipulation under ether anesthesia and another cast applied. He suffered severe pain in the back after this procedure for three days but it subsided subsequently. This cast, worn for four weeks, produced great improvement in his posture, an improvement he has maintained since that time. There has been no pain whatever for the last ten years and his health has been good.

Before onset of his illness this man weighed 175 pounds and was 6 feet 2 inches tall. He now weighs 155 pounds.

The most interesting features of the physical examination are shown in the photograph (Fig. 1). The posture is characteristic of the disease with moderate kyphosis of the dorsal spine, forward inclination of the cervical spine and hyperextension of the head. The spine is completely stiff throughout its entire length and consequently its shape is not changed at all when the patient stoops. He is able to bend over by hip motion alone. The lumbar spine is flattened obliterating the normal lordosis. Chest expansion is nil. The range of head motion is not restricted. The gait is awkward, a sort of waddle, characteristic of this disease. All other joints are normal.

Roentgen Findings. Anteroposterior and lateral projections of the lumbosacral spine and pelvis and lateral projections of the cervical

and thoracic vertebrae showed a diffuse "washed-out" appearance of the vertebral bodies, the result of severe generalized bone atrophy. The anterior, posterior and lateral spinal ligaments were calcified, forming a definite bridging between the bodies of the cervical and lumbar vertebrae producing the so-called "bamboo spine." A moderate degree of cupping of the upper and lower end-plates of the vertebral bodies were present indicating expansion of the cartilaginous discs, the result of softening of the vertebral bodies due to demineralization. These changes were less pronounced in the thoracic region where a moderate degree of kyphosis existed. The majority of the small apophyseal joints were obliterated by healing changes in the form of bony ankylosis. Both sacroiliac joints showed a combination of sclerotic and atrophic changes with complete disappearance of any evidence of these joints ever having been present. An additional feature, not observed regularly in ankylosing spondylitis, was the presence of amorphous-like calcifications in several of the disc cartilages in the thoracic and lumbar regions. The hip joints were of normal appearance (Fig. 3*A*, 4*A* and 5).

Paul has always been able to work except for the periods of hospitalization as previously described. He is now employed as a machinist.

Case II. Reuben is now forty-seven years of age. Arthritis began gradually at the age of twenty-nine years as pain in one hip spreading gradually to the other hip and involving the lower back about three years after onset. Nine years after onset, the patient was hospitalized and kept in bed in hyperextension for three months. This resulted in marked improvement in his posture. He wore a cast for nine months after discharge from the hospital and later a back brace. Before he became ill he weighed 200 pounds and was 6 feet 2 inches tall. Reuben was unemployed for several years only because of the depression, not because he was too sick to work. He is now quite well and works regularly as an elevator maintenance man.

Physical examination is about the same as that described for Paul except that the kyphosis is not so marked. The lack of mobility, the absence of chest expansion, and normal function of peripheral joints are the same in both cases (Fig. 1 and 2). He now weighs 155 pounds.

Roentgen Findings. Anteroposterior and lateral projections of the lumbar spine and pelvis presented changes in the sacroiliac joints similar to those found in Case I. The calcification in the spinal ligaments was less marked and the apophyseal joints were only moderately involved (Fig. 3*B* and 4*B*). In the thoracic segment there was even less marked involvement of the apophyseal joints and ligamentous structures. The cervical spine was free of disease except for large osteophytes on the lower margin of the body of the fifth and upper margin of the body of the sixth vertebrae. Comparison of roentgenograms of the lumbar spine and pelvis made at a ten year interval disclosed only an increase in the amount and density of the calcification in the spinal ligaments and apophyseal joints.

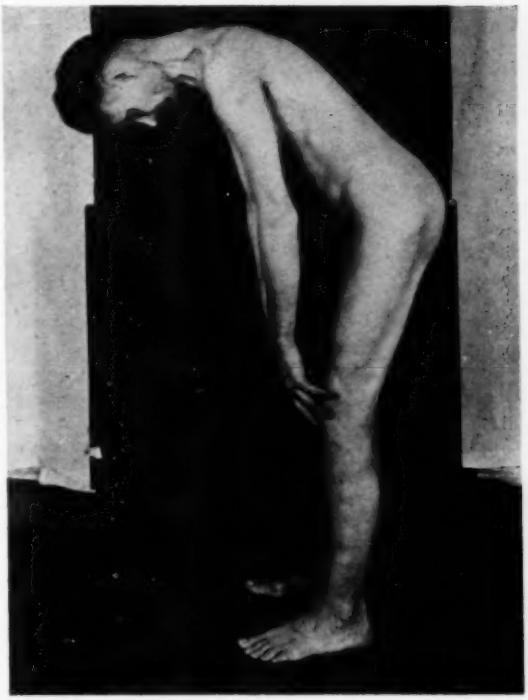

Fig. 2. Photograph of Case II showing immobility of the lumbar and dorsal spine. Flexion occurs at the hips and in the cervical spine.

LITERATURE

Weil and Allolio[9] described ankylosing spondylitis in brothers, two cases quite similar to the ones described above. In Peter, the older brother, the disease began as mild backache at the age of twenty-two;

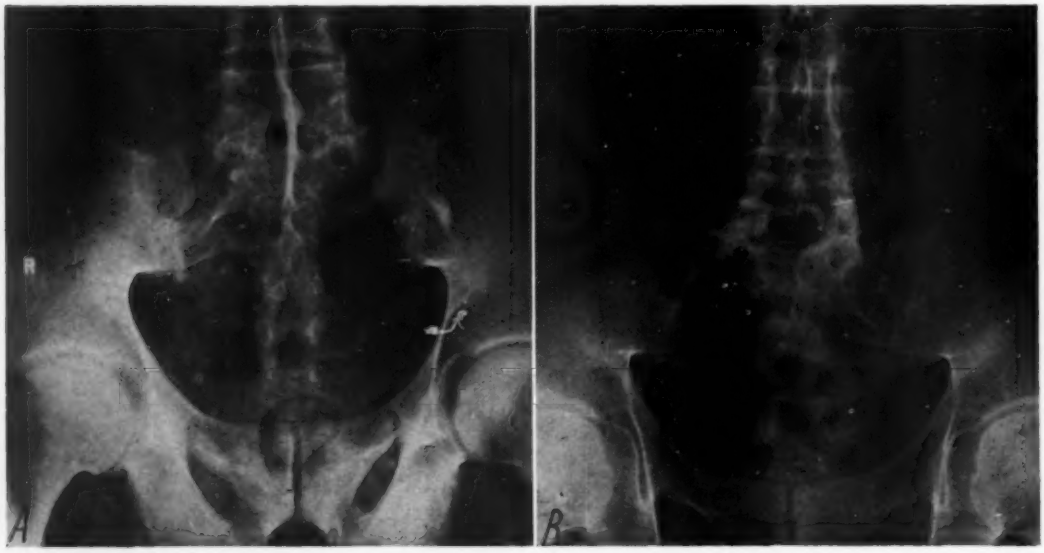

FIG. 3. *A*, Case I. Calcification and ossification of lumbar spinal ligaments, ankylosis of the apophyseal joints and obliterations of the sacroiliac joints. *B*, Case II. Lumbar spine and pelvis showing calcification of the apophyseal joints and spinal ligaments. The sacroiliac joints are completely ankylosed.

it became so severe in two years that the patient was practically bedridden. The following year he had sciatica in both legs and a constant pain extending farther up his back. His gait became slow and staggering, and he developed a marked kyphosis. The pain became more severe and he gave up work entirely ten years after onset.

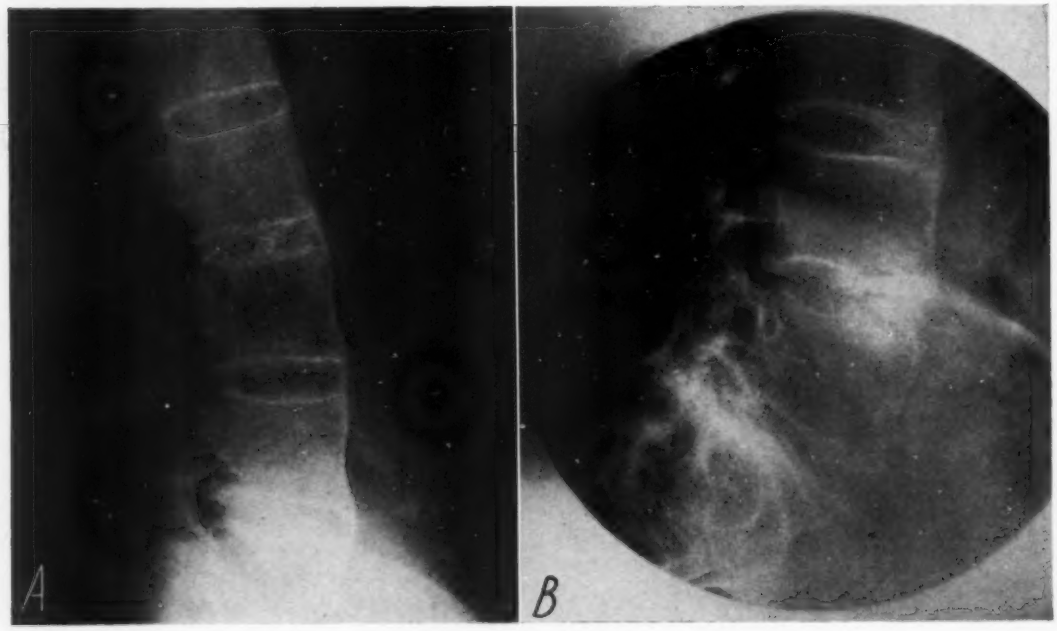

FIG. 4. *A*, Case I. Lateral projection of lumbar spine showing generalized atrophy, cupping of end-plates and calcification of the anterior ligament and second intervertebral disc. *B*, Case II. Lateral view of lower lumbar spine shows atrophy of the bodies, calcification of the anterior ligament, cupping of the end-plates and amorphous calcification in the disc cartilages.

Physical examination showed that the entire back was completely stiff up to the occiput. There was kyphosis of the thoracic region and flattening of the lumbar spine. The peripheral joints including hips and shoulders were completely normal. Roentgenological examination showed ossification of the interspinous ligaments with spurs in lumbar and lower dorsal region.

Mathias, the second brother, noted pain in the hips, sacrum and lower back coming on at the age of nineteen after he froze his feet. The pain was aggravated by standing. Since the age of twenty-six the pain has been constant; he had bowing of the back and pain and stiffness in the shoulders and the neck. Physical examination showed the spine to be completely stiff; there was kyphosis in the dorsal region and flattening of the lumbar spine. Roentgenological examination showed beginning calcification of the interspinous ligaments.

The authors believe these are typical cases of ankylosing spondylitis. A photograph of both brothers shows that both have the typical posture of the disease. In both instances the diseases began after exposure to cold and were thought to have been related to war service.

Reynolds[8] reported another instance of the disease in brothers. Robert M., aged twenty-eight, a soldier in the Army, appeared normal except for some stiffness of the back. Roentgenograms showed changes about the sacroiliac joints with demineralization and beginning lipping in the dorsolumbar spine which warranted a diagnosis of Marie-Strümpell type of spondylitis. His brother, James M., aged twenty-six, also had stiffness of the spine. Roentgenograms taken in 1943 showed obliteration of sacroiliac joint spaces with early lipping of the cervical and lower dorsal spine, changes of Marie-Strümpell spondylitis. Roentgenograms repeated a year later were essentially the same.

Ehrlich in an extensive review of 753 cases noted the occurrence once of ankylosing spondylitis in brothers. He gives no details except to state that one of the brothers was discharged from the Army because of his condition.

Fig. 5. Case 1. Calcification in the anterior and posterior cervical spinal ligaments, atrophy and cupping of the bodies, and obliteration of the apophyseal joints.

Other instances have been found in the literature of multiple cases of ankylosing spondylitis in one family but the evidence is not convincing. Herrick and Tyson[4] describe a thirty-two year old man with complete fusion of the sacroiliac joints and obliteration of the apophyseal joints of the thoracic and the lumbar spine. He had had symptoms for six years. His father was said to have had a poker spine. Blair[1] described a thirty-two year old man with demineralization of the spine, fusion of the sacroiliac joints, deformity of the pelvis and ankylosis of the spine. The diagnosis of ankylosing spondylitis would seem to be admissible here but it is in reasonable doubt concerning the twenty-three old brother. The latter patient had been disabled twice because of trauma to the back. Examination showed some stiffness of the spine, muscle spasm and difficulty with leg raising. "Roentgenograms showed irregularity of the sacroiliac joints with sclerosis of the adjacent bone and some decrease in definite markings of the lower lumbar postero-lateral

articulations." The diagnosis of ankylosing spondylitis would seem to be admissible in the first case but is doubtful in the second.

Weil and Allolio mentioned two further instances of familial involvement of the spine.

Marie and Astie[6] described an old man and his sister with senile kyphosis. Magnus-Levy[5] described another man with senile kyphosis who had two maternal aunts similarly affected.

DISCUSSION

We have here described the occurrence of ankylosing spondylitis in brothers. We have mentioned a second example of this phenomenon from personal communication and have described two additional instances from the literature which seem to be authentic. The cases of Herrick and Tyson involved a man and his father but the latter was not examined and so the diagnosis was not reliably established. The second brother of Blair's set is not acceptable. Senile kyphosis is quite a different disease from ankylosing spondylitis. It is a disease of old age; it is a result of severe demineralization and is characterized by marked dorsal kyphosis. The intervertebral discs become thin anteriorly so that fusion of vertebral bodies occurs in front. The apophyseal and sacroiliac joints are not involved.

The discovery of four instances of ankylosing spondylitis affecting brothers may be of no etiological significance. The disease is not rare so that multiple cases in the same sibship can be expected to occur occasionally by chance alone. The likelihood of such an occurrence resulting from chance alone cannot be computed from any statistics on the subject known to the authors. It may be relevant to note that although it is much more common than ankylosing spondylitis, we know of only one instance of family involvement of rheumatoid arthritis, in which it affected sisters. Four instances of ankylosing spondylitis affecting brothers suggests a hereditary factor in this disease. We know of no other evidence supporting this proposition. Attempts have been made to explain the etiology of spondylitis on constitutional factors. The individuals herewith presented resembled each other closely in size, body build, pigmentation and facial features.

We are forced to conclude that the occurrence four times of ankylosing spondylitis in brothers does not lead to an explanation as to etiology because such a coincidence may have resulted from chance alone.

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OSSIFICATION OF THE CORACOCLAVICULAR LIGAMENT FOLLOWING DISLOCATION OF THE ACROMIOCLAVICULAR ARTICULATION

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CALCIFICATION or ossification of ligaments and tendons following trauma is recognized as a common condition and, in certain locations, is the usual sequela of hemorrhage into the injured structures. Codman[2] in his classic work on the shoulder described the type of calcification that is noted in the supraspinatus and other tendons following injury. Ossification frequently occurs in the medial collateral ligament of the knee and in hematomata beneath the quadriceps femoris, within the brachialis anticus, the rectus abdominalis and other muscles. Following injury to certain joints, notably the elbow, knee and ankle, small particles of osseous material appear in the peri-articular tissues weeks or months following injury.

Ossification of the coracoclavicular ligament following partial or complete avulsion of this ligament incidental to dislocation of the acromioclavicular articulation is a less well known condition, in spite of the fact that it is apparently a common sequela of a relatively common type of injury. In the series reported below, of 18 patients with dislocation of the acromioclavicular articulation, 14 subsequently developed osseous deposits in the coracoclavicular ligament.

ANATOMICAL FACTORS

The acromioclavicular articulation is a diarthrodial joint between the center of the medial border of the acromial process of the scapula and the acromial end of the clavicle. The strength of the joint is largely in the ligaments about it—the articular capsule, the superior and inferior acromioclavicular and coracoclavicular ligaments. The articular capsule encases the joint, being attached about the articular margins. It is augmented above by a dense ligamentous band, the superior acromioclavicular ligament, which is continuous with fibers of the aponeuroses of the deltoid and trapezius muscles. Beneath the capsule is a similar band, the inferior acromioclavicular ligament.

The coracoclavicular ligament, while not in continuity with the capsule of the acromioclavicular articulation, is of great importance in maintaining the normal relationship of the clavicle to the acromion process. This ligament consists of two fasciculi—the conoid and trapezoid ligaments (Fig. 1). The trapezoid fasciculus is a thin band of tough fibrous tissue which extends between the superior surface of the coracoid process and the oblique ridge on the inferior surface of the lateral third of the clavicle. The conoid fasciculus is a cone-shaped band, the apex of which is attached to the base of the coracoid process, the base to the conoid tubercle on the inferior surface of the clavicle. These two fasciculi join one another at an angle directed posteriorly.

The coracoclavicular ligament thus anchors the clavicle securely to the coracoid process. Rotation of the scapula on the acromial end of the clavicle is limited by the two fasciculi—the trapezoid ligament limiting forward rotation and the conoid ligament limiting backward rotation.

Bosworth[1] states that the coracoclavicular ligament may be likened to a stout cord by which the scapula and with it the whole upper extremity is suspended from the outer end of the clavicle.

Authorities differ regarding the function of the clavicle. The opinion which seems to be most commonly accepted is expressed by Mumford[4] who states that the function of the clavicle, other than for attachment and origin of certain muscles, is purely one of supplying a strut or buttress to hold the tip of the shoulder out from the body. There is abundant evidence that defects in or absence of the clavicle do not produce disability. In mutational (cleidocranial) dysostosis, the clavicles are usually either absent or defective and shoulder function

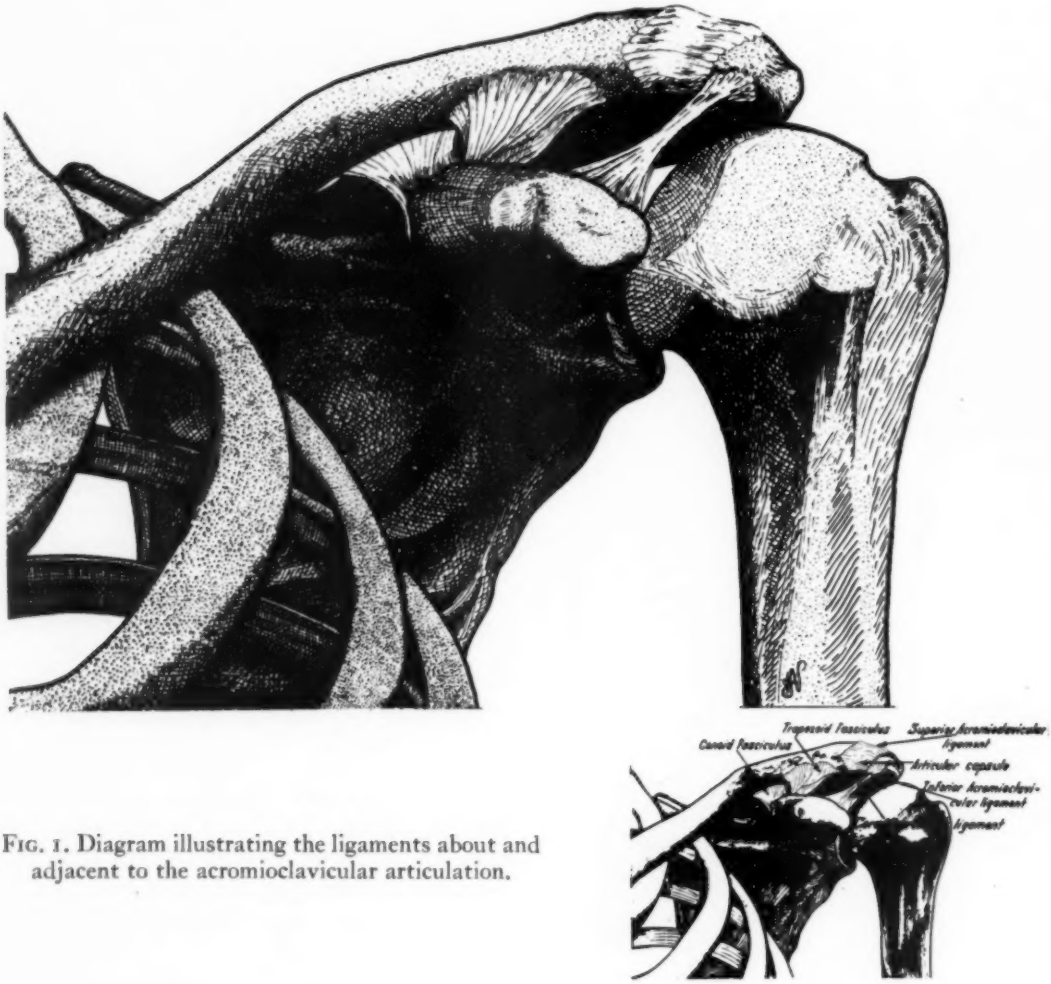

Fig. 1. Diagram illustrating the ligaments about and adjacent to the acromioclavicular articulation.

in such patients is rarely if ever abnormal. There are several cases on record where the entire clavicle was removed without subsequent disability. It seems probable in acromioclavicular separations that part of the disability which frequently follows such injuries comes from the fact that other structures of the shoulder have dropped away from the clavicle and the elevated acromial end of the clavicle pushes against the overlying soft tissues in certain motions. If several centimeters of the acromial end of the clavicle are resected, function is usually restored.

DISLOCATIONS OF THE ACROMIOCLAVICULAR ARTICULATION

Dislocations of this joint are described as incomplete or complete, the latter term being applied when the coracoclavicular ligament is severed. In all dislocations, there is tearing of the articular capsule and usually of the superior or inferior acromioclavicular ligaments or both.

The commonest causes of either complete or incomplete dislocations are blows or falls directly upon the shoulder. Sudden force drives the scapula downward and backward. Downward excursion of the clavicle is limited by the rigid first rib below and by muscle pull from above with consequent tearing of the ligaments between the clavicle and scapula. Frequently, torn bits of capsule and ligaments become interposed between the clavicle and acromion, thus preventing complete reduction of the dislocation.

TABLE I

TABULATION OF CASES OF ACROMIOCLAVICULAR DISLOCATIONS

Case No.	Age	Date of Injury	Last Examination Before Ossification	First Examination After Ossification	Time Interval	Type of Injury	Clinical Notes	Result at Last Observation
I	26	6-18-43	6-22-43	7-10-43	More than 4; less than 22 days	Injured while wrestling	See case summary	Asymptomatic, 5 months
II	35	5-27-44	6-1-44	6-26-44	More than 5; less than 30 days	Fell in scuffle	See case summary	Improved, 2 months
III	37	6-3-43	6-3-43	1-21-44	Less than 8 months	Injured while wrestling	See case summary	Unimproved, 8 months
IV	30	3-31-44	3-31-44	5-30-44	Less than 60 days	Injured in train wreck	See case summary	Asymptomatic, 3 months
V	29	1-4-43	1-4-43	5-30-43	Less than 150 days	Thrown from jeep	See case summary	Improved, 11 months
VI	37	3-4-42	3-4-42	10-13-43	Less than 19 months	Fell against railroad track	See case summary	Unimproved, 2 years
VII	25	11-8-43	12-27-43	—	—	Fell, hitting side of building	See case summary	Asymptomatic, 2 months
VIII	37	7-7-42	12-19-42	—	—	Pulled shoulder in obstacle course	Resection of acromial end of clavicle; no ossification within 5 months	Improved, 6 months
IX	35	8-5-42	1-23-43	—	—	Fell while wrestling	Incomplete disolocation. No ossification within 6 months	Asymptomatic, 6 months
X	32	2-25-43	3-11-43	3-31-43	More than 14; less than 34 days	Thrown on shoulder; judo	Complete dislocation treated by operation (sutures of wire and fascia). Osseous deposits in ligament	Improved, 3 months
XI	50	1935	—	3-22-43	Less than 8 years	Horse fell on shoulder	Patient studied 8 years after injury; limitation of motion and history of intermittent pain; osseous deposits in ligament	No change
XII	33	6-3-43	6-3-43	8-7-43	Less than 65 days	Injured in jeep accident	Wide separation of joint treated conservatively; small amount of ossification in ligament	Improved, 5 months
XIII	38	7-8-43	7-22-43	9-1-43	More than 14; less than 54 days	Fell from truck, landing on shoulder	Moderate separation of joint; small amount of ossification	Improved, 4 months
XIV	27	12-30-43	—	4-8-44	Less than 100 days	Thrown from vehicle	Slight disability 4 months after injury; moderate separation of joint and partial ossification of ligament	Improved, 6 months
XV	45	1940	—	4-13-44	Less than 4 years	Auto accident	Slight disability with intermittent pain; moderate separation of joint and partial ossification of ligament	Unimproved, 4 years
XVI	23	6-26-44	8-26-44	—	—	Injured, playing football	Clinically incomplete separation treated by strapping; no ossification within 2 months	Asymptomatic, 2 months
XVII	29	8-16-43	—	8-23-44	Less than 1 year	Injured in fall, landing on shoulder	Clinically complete separation treated conservatively; 1 year later, small osseous masses in ligament	Improved, 1 year
XVIII	34	11-21-44	12-7-44	12-28-44	More than 16; less than 37 days	Caught between two trucks	Sustained bilateral dislocations of acromioclavicular joints; 2 months later no disability; moderate ossification of both coracoclavicular ligaments	Asymptomatic, 2 months

Speed[6] states that while there may be associated damage to muscles and fascia, there is little blood extravasation present and injuries of blood vessels and nerves are almost unknown. Small fragments of bone and cartilage may be detached from the clavicle or acromion and occasionally a fracture or dislocation may be present in the shoulder. He also states that after rupture of the fasciculi of the coracoclavicu-

lar ligament a process of calcification may infiltrate them and aid in stiffening the shoulder region. He believes that this ossification reaction undoubtedly comes from a tearing of the periosteum and the irritation from too early movement and use of the joint.

Liberson[3] states that post-traumatic ossification of the ligaments, resulting either from injury or from improper attention to the acromioclavicular dislocation with injury to the ligaments, causes considerable hardship and disturbed function of the shoulder girdle.

Murray,[5] on the other hand, reports several cases of acromioclavicular dislocation treated by insertion of Kirschner wires through the outer end of the clavicle and acromial process of the scapula in which there was development of extensive calcification of the coracoclavicular ligaments without subsequent deformity or disability.

If unsuccessfully reduced, the principal late effects of such dislocations are weakness and partial disability of the shoulder girdle, especially in such motions as lifting, pushing or picking up objects. The clavicle is virtually divorced from the remainder of the shoulder girdle and force which is usually transmitted to the trunk through the scapula and clavicle and their muscular and ligamentous attachments is thrown out of balance. This may amount to complete disability in a manual laborer

ROENTGEN FINDINGS

Anteroposterior roentgenograms of both shoulders taken in the upright position with a 20 pound weight in each hand will demonstrate widening of the injured joint and elevation of the acromial end of the clavicle. Small splinters of bone may be demonstrated if they are detached from the clavicle or acromion process. There is variability in the width of the joint space in different individuals; occasionally spaces as wide as 1 cm. may be present without dislocation. However, the disproportionate width of the injured joint and the abnormally high position of the acromial end of the clavicle are the important diagnostic features.

Ossification of the coracoclavicular ligament has been observed as early as twenty-two days following injury. It usually appears as a group of amorphous, cloudy areas of light density below the outer third of the clavicle, frequently near the conoid tubercle and usually closer to the clavicle than to the coracoid process. These areas increase rapidly in size and density and within several weeks take on definite characteristics of bone, this being laid down in strands and spicules corresponding more or less to the lines of the fibrous strands of the conoid and trapezoid fasciculi. While the bone frequently becomes attached firmly to the clavicle above, it approaches, but rarely becomes continuous with the coracoid process below. The process of ossification appears to be progressive for a period of about eight to ten weeks and then shows no perceptible change on subsequent examinations.

Speed states that post-traumatic calcification is apt to be less marked if the shoulder is immobilized promptly. In the cases reported below, the production or absence of ossification appeared to have no relationship to motion as nearly all cases were immobilized shortly after injury.

If ossification has not appeared within six weeks following injury, it has not been noted in subsequent examinations.

It should be emphasized that while several authors have used the term "calcification" to refer to the changes present in the coracoclavicular ligament, the process actually is one of ossification with material easily recognizable as bone being laid down within the substance of the ligament.

REPORT OF CASES

Case 1. A corporal, aged twenty-six, sustained a complete separation of the right acromioclavicular articulation while wrestling. A Velpeau bandage was applied within a few minutes following injury, but the deformity and pain persisted. Five weeks later, an open

reduction was performed and the clavicle and acromion process were approximated and held by fascial strips and a loop of wire was inserted through drill holes in each. Two months later the patient was sent to a rehabilitation center where he remained for six more weeks before being returned to full duty. Upon discharge, he had full range of motion and no pain or other complaints.

Roentgenograms of the right shoulder taken four days following injury (Fig. 2) shows the separation of the acromioclavicular articulation. Figure 3, a roentgenogram taken twenty-two days following injury, shows a moderate amount of amorphous, cloudlike material in the soft tissues beneath the outer end of the clavicle and persistence of separation of the joint with elevation of the clavicle. Forty-eight days after injury (Fig. 4), the soft tissue deposits are larger and denser, but the bone is still somewhat amorphous in character. Figures 5 and 6 are roentgenograms with and without weight bearing taken ninety-one days following injury. Well developed osseous masses are now noted in both fasciculi of the coracoclavicular ligament. On weight bearing, there is still slight elevation of the acromial end of the clavicle, but very little increased widening of the space be-

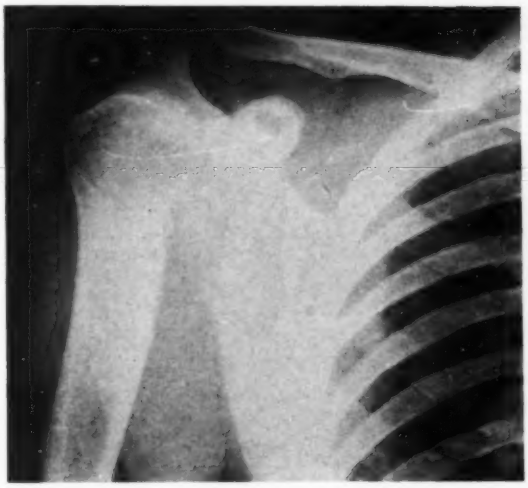

Fig. 2. Case 1. Four days following injury.

Fig. 3. Case 1. Twenty-two days following injury. Early signs of ossification are appearing in the conoid and trapezoid fasciculi.

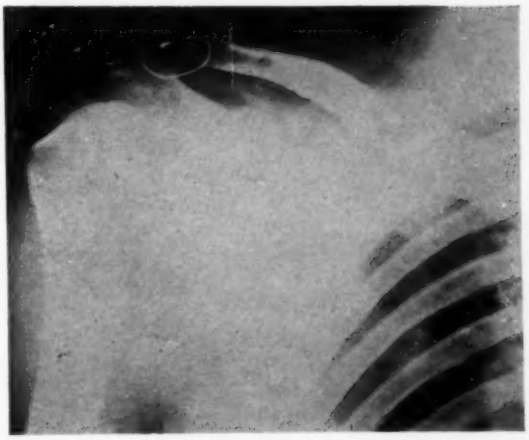

Fig. 4. Case 1. Forty-eight days following injury. More extensive ossification has appeared but this is still more or less amorphous in character.

Fig. 5. Case 1. Ninety days after injury. Ossification is now complete.

Fig. 6. Case 1. Weight bearing. The acromioclavicular articulation is still lax but patient was asymptomatic.

tween the coracoid process and the clavicle. Subsequent roentgenograms showed no further change in the amount or appearance of the osseous deposits.

Case II. This soldier, aged thirty-five, while on guard duty attempted to separate two soldiers who were fighting in the barracks. While so doing, he fell with one soldier on top of him, sustaining a complete dislocation of the right acromioclavicular articulation. His arm was immobilized in complete abduction for forty-eight hours and was then strapped in an attempt to reduce the separation. Figure 7 is a roentgenogram taken on the day of injury showing separation of the joint and elevation

Fig. 7. Case II. On day of injury. Roentgenogram demonstrates slight separation of the joint and elevation of the clavicle.

Fig. 8. Case II. Thirty days after injury. Amorphous ossification has appeared in both fasciculi of the coracoclavicular ligament.

of the acromial end of the clavicle. Figure 8 is a roentgenogram taken thirty days after injury. Rather abundant amorphous ossification has already appeared in both fasciculi of the coracoclavicular ligament. Figure 9 is a roentgenogram taken fifty-nine days after injury with the patient upright and bearing a 20 pound weight in his right hand. The acromial end of the clavicle is still elevated; ossification is now complete and while it is continuous with the clavicle above, there is a narrow space between the ossified portions of the ligament and the coracoid process.

At the time of this examination, the patient had no complaints, but experienced slight difficulty in lifting heavy objects.

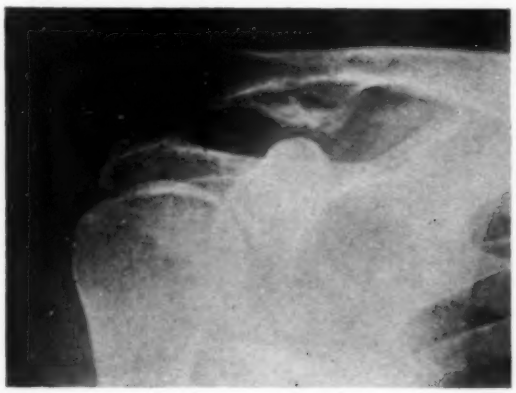

Fig. 9. Case II. Fifty-nine days after injury with patient holding a 20 pound weight. Ossification is now complete; a narrow space exists between the ossified portions of the fasciculi and the coracoid process.

Case III. A lieutenant, aged thirty-seven, was studied eight months following injury sustained while wrestling when he was thrown heavily, landing on his left shoulder. Roentgenogram on the day of injury (Fig. 10) shows separation of the joint but no elevation of the clavicle. He carried his arm in a sling for several weeks but his shoulder was not immobilized. During the next few months he had intermittent attacks of pain in the shoulder and when examined eight months following injury was unable to elevate his arm above horizontal. Roentgenography in the recumbent position (Fig. 11) showed marked ossification of the conoid fasciculus and slight ossification of the superolateral portion of the trapezoid fasciculus. Roentgenography in the upright position with a 20 pound weight in the right hand (Fig. 12) shows widening of the joint space, but depression of the clavicle, indicating at least partial restoration of continuity of the coracoclavicular ligament. Roentgenography in abduction (Fig. 13) shows attachment of the osseous mass to the clavicle but not to the coracoid process.

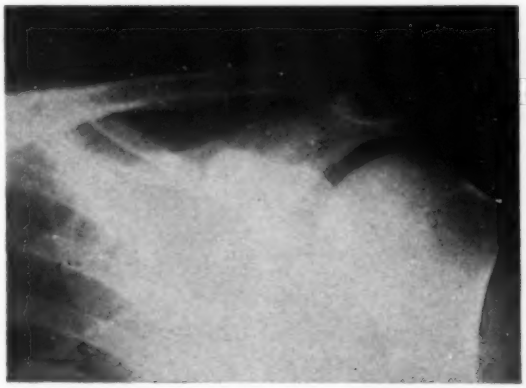

Fig. 10. Case III. On day of injury. There is moderate widening of the joint space but only slight elevation of the clavicle.

Fig. 11. Case III. Eight months after injury with patient recumbent. There is marked ossification of the conoid fasciculus and slight ossification of the superolateral portion of the trapezoid fasciculus.

Fig. 12. Case III, same date. Patient is upright, bearing 20 pound weight in his hand. While the joint space widens, the clavicle is depressed, indicating at least partial restoration of continuity of the coracoclavicular ligament.

Fig. 13. Case III, same date. Left shoulder in abduction. Osseous masses are attached to the clavicle but not to the coracoid process.

Case IV. This infantryman, aged thirty, was injured in a train wreck, sustaining a dislocation of his right acromioclavicular articulation, a fracture-dislocation of the cervical spine without paralysis and comminuted fractures of left tarsal bones. His shoulder was strapped for one month. When first seen in this hospital three

Fig. 14. Case iv. Three months after injury. Roentgenogram in recumbent position.

months later, he had no symptoms as far as his shoulder was concerned and full range of motion was present. At this time, roentgenograms in recumbent position (Fig. 14) and upright bearing a weight (Fig. 15) demonstrated bands of ossification in both fasciculi of the coracoclavicular ligament with attachment to the clavicle and coracoid process.

Case v. A lieutenant, aged twenty-nine, was studied eleven months following an accident when he was thrown about 40 feet from a jeep, landing on his right shoulder. Roentgen examination of the shoulder revealed moderate separation of the acromioclavicular articulation. His shoulder was taped for four weeks and he then carried his arm in a sling for a few additional days. Three months following the accident, roentgenograms showed extensive ossification of the coracoclavicular ligament and slight separation of the acromioclavicular articulation. At the time of examination, eleven months after injury, he had no complaints other than a feeling of slight weakness in the shoulder on pulling or lifting heavy objects. There was no limitation of motion and no tenderness. Roentgenography of the shoulder at this time (Fig. 16) showed discrete osseous deposits in both fasciculi of the coracoclavicular ligament but no separation of the joint or elevation of the clavicle. It seems probable that the complete recovery in this case may be credited, in part at least, to the ossification of the ligament.

Fig. 15. Case iv. Patient upright and bearing 20 pound weight. Bands of ossification are noted in both fasciculi; the joint space is slightly wide but the clavicle is only slightly elevated. Patient was asymptomatic at this time.

Case vi. This soldier, aged thirty-seven, had sustained a complete dislocation of the right acromioclavicular articulation in civilian life two years before, when he had slipped and

Fig. 16. Case v. Eleven months following injury. There are well developed osseous masses in the coracoclavicular ligament but there is no separation of the acromioclavicuar articulation.

fallen, striking his shoulder against a railroad track. He was admitted to the hospital with a history of persistent pain in his right shoulder and inability to use his shoulder in such motions as lifting, pushing and pulling. Examination revealed moderate separation of the right acromioclavicular articulation with crepitus and painful motion of the shoulder joint. Roentgenogram of the right shoulder (Fig. 17) showed separation of the acromioclavicular articulation and osseous masses in both fasciculi of the coracoclavicular ligament. The patient was discharged from the service.

Case vii. This soldier, aged twenty-five,

slipped and fell from a step while carrying a box, striking his shoulder against a building. He experienced considerable pain in the region of the acromioclavicular joint. Roentgenogram of the shoulder with the patient recumbent revealed no abnormality, but roentgenogram in the upright position with a 20 pound weight in his hand (Fig. 18) showed slight separation of the joint and slight elevation of the acromial end of the clavicle. The dislocation was treated by adhesive strapping. Symptoms rapidly disappeared and he was returned to full duty two months after injury. Roentgenograms taken at this time showed no evidence of subclavicular ossification. This apparently represents a case of incomplete dislocation without avulsion of the coracoclavicular ligaments.

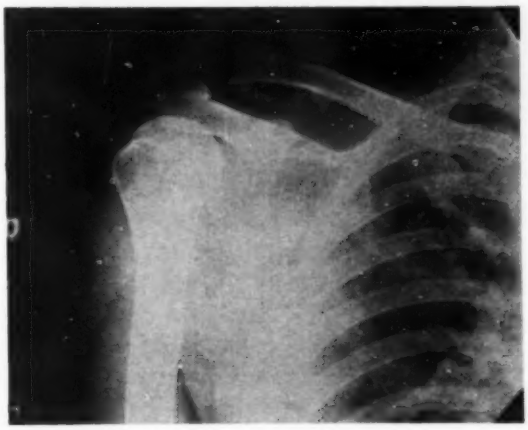

Fig. 17. Case vi. Two years following injury. Roentgenogram demonstrates slight widening of the joint space, slight elevation of the clavicle and multiple osseous masses in the coracoclavicular ligament.

Fig. 18. Case vii. Several days following injury. Roentgenogram taken in the upright position, the patient holding a 20 pound weight, shows slight separation of the joint and slight elevation of the clavicle. This patient did not develop ossification of the ligament and the dislocation was classified as incomplete.

SUMMARY

1. Post-traumatic ossification of the coracoclavicular ligament is a common sequela of avulsion of this ligament associated with dislocation of the acromioclavicular articulation.

2. It is seen more commonly in dislocations which are classified clinically as complete.

3. The ossification appears three to six weeks following injury and becomes complete about eight to ten weeks following injury.

4. If ossification has not appeared within six weeks following injury, it probably will not appear at a later date.

5. The ossification does not appear to contribute to the disability of patients, but may aid in restoring continuity of the damaged ligaments and in stabilizing the acromioclavicular articulation.

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A STUDY OF LOWER EXTREMITY LENGTH INEQUALITY

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THE observations to be presented are those made by the study of 1,000 lumbosacral spines of soldiers examined in the X-Ray Department of the Regional Station Hospital of Fort Leonard Wood, Missouri. Most significant is the fact that the method of roentgenographic examination is unique, in that it institutes the use of a special machine devised by one of us (W.A.R.), and by the use of this machine all spine studies have been made in an absolutely standardized upright position. An illustration of this device is seen in Figure 1. This device has been described and illustrated in a paper[1] recently published.

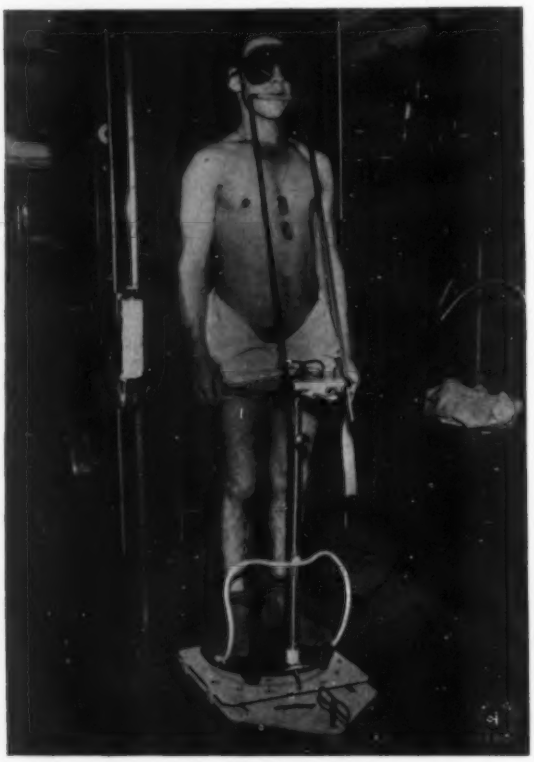

FIG. 1. The spinal fixation and stabilization device with patient in position for the anteroposterior roentgenogram.

The instrument is known as the spinal fixation and stabilization device. The roentgen-ray table is placed in the vertical position and the machine is brought up against it as seen in the illustration. The patient to be examined mounts the rotatable platform, standing on it in his stocking feet. He then is instructed to adjust a movable, centrally placed saddle so that it fits snugly into his crotch, thus assuring that he is centrally placed on the platform. An additional adjustable stabilization bracket is used to insure that the patient will stand as erect as is comfortably possible. This is accomplished by a bracket brought up to the patient's mouth, and on this bracket sterilized rubber tubing is placed and the patient is instructed to bite the tubing. The bracket is then pushed up into the position which holds the patient in an erect posture and it is then locked. The technician inspects the patient to be certain that both knees are held in the position of complete extension. The rotatable platform is then adjusted and locked so that the patient stands in an absolute anteroposterior presentation and the roentgenogram is made, using a 14 by 17 film size, so that the film will include the heads of both femurs, the entire lumbar and sacral spine, and a portion of the lower dorsal spine will be seen on the upper part of the roentgenogram. Then without moving, the patient is rotated by means of the rotatable platform so that it now is turned at 90 degrees and the patient is then presented for a true lateral view. A second, that is, lateral, roentgenogram is made in this position. This completes the roentgenographic study of the patient. As described in the original paper, it is again mentioned here that studies may be accomplished at any desired

angle, according to the set of the rotatable platform, and by this means it is possible to visualize foramina, pedicles, articular facets, and any other portion of the spine best seen in the oblique views. By knowing such angles these views can be reproduced at any subsequent examinations. However, in this study, only the anteroposterior and lateral views were used.

By measurements and special tests it was determined that the lower edge, or border of a film taken in the upright position is absolutely parallel to the plane of the platform on which the patient stands. Accordingly, when the anteroposterior roentgenogram is examined, any difference in the level of the femoral heads as determined by measuring from the lower edge of the film to the uppermost portion of the femoral heads, may be taken to indicate a difference in lower extremity lengths. In addition to the determination that the lower border of the film is absolutely parallel to the plane of the platform of the stabilizing machine, a series of exposures were made on the same patient under variable conditions to determine possible sources of error. The patient was first examined as was routinely accomplished in this series, that is, the normal upright anteroposterior position. Then, while still standing in the anteroposterior position, he was instructed to attempt to tilt his pelvis by straining to one side and at the same time keeping both heels firmly on the platform. An additional exposure was made by repeating this procedure in which the patient attempted to tilt his pelvis to the opposite side. These roentgenograms were then examined and, by measurement, no differences were demonstrable. This would substantiate the absolute fixation of the stabilization device, and accordingly it is felt that the measurement which had been made may be accepted as reasonably accurate. In Figure 2, anteroposterior roentgenograms of three different patients are illustrated. *A* is one in which the femoral heads are of equal height when measured from the lower border of the film, and accordingly the right and left lower extremities on this patient are assumed to be of equal length. *B* illustrates a patient in whom the right lower extremity is shorter than the left as seen by mensuration, and *C* is of a patient in whom the left leg is shorter than the right.

The soldiers used in this study represent 1,000 consecutive, non-selected cases who were sent to the roentgen department because of a low back complaint and in every instance the roentgenograms were made by the use of the fixation device referred to above. In this study, all of the roentgenograms were reviewed first for the purpose of mensuration to determine the equality or amount of inequality between the lower extremities. All of the roentgenograms were then reviewed a second time for the purpose of determining existing pathology as seen roentgenographically. Each case was referred to by number in order that the result of each of these two reviews of the roentgenograms could be correlated. Tabulation of measurement as done in the first review and of roentgenographic pathology as done in the second review was carried out entirely separately in order that there would be no subconscious attempt on our part to correlate pathology with difference in extremity measurements. Only after the tabulations were completed was a correlation of the results made.

In tabulating what we refer to as leg length, the actual amount of difference between the height of the femoral heads is measured in millimeters. Tabulation was made on the basis of: (1) the right leg being shorter than the left, designating the amount, and (2) the left leg being shorter than the right, again indicating the amount.

For a reason, to be indicated later in this study, measurements were grouped on the basis of being 5 mm. or less and exceeding 5 mm. in amount. Differences as slight as 1 mm. were recorded. In one instance, a right lower extremity measured 38 mm. shorter than the left, and in another case a left lower extremity measured 44 mm. shorter than the right. These 2 cases represent the greatest extremes which were observed.

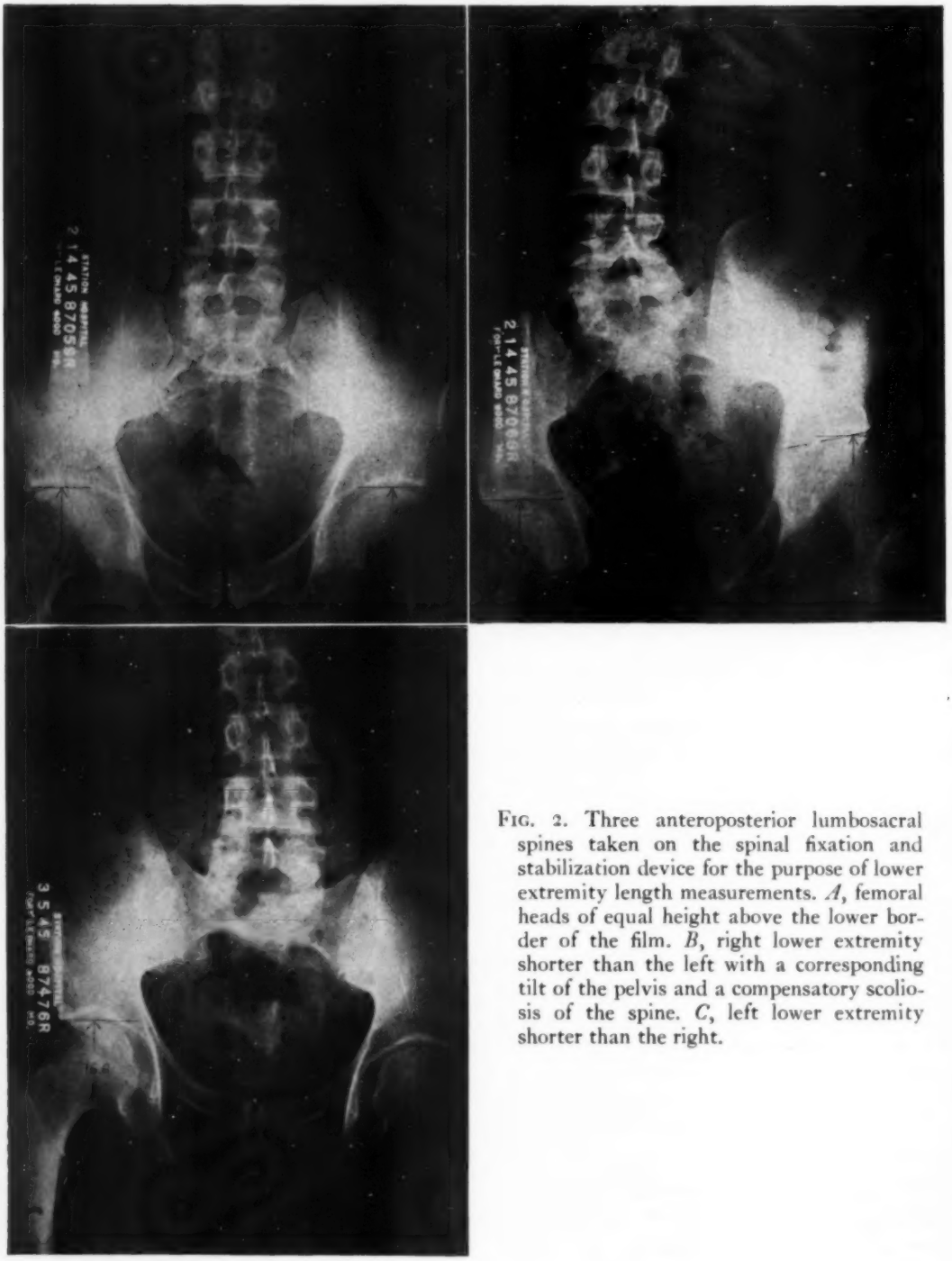

FIG. 2. Three anteroposterior lumbosacral spines taken on the spinal fixation and stabilization device for the purpose of lower extremity length measurements. *A*, femoral heads of equal height above the lower border of the film. *B*, right lower extremity shorter than the left with a corresponding tilt of the pelvis and a compensatory scoliosis of the spine. *C*, left lower extremity shorter than the right.

Table 1 gives the measurements obtained in the 1,000 cases reviewed. Only 230, or 23 per cent, of the cases had lower extremities of equal length. Of the 406 cases in which the right lower extremity is shorter than the left, the average shortening in these 406 cases was found to be 7.47 mm. Of the remaining 364 cases which manifested shortening of the left lower extremity, the average shortening was found to be 6.50

mm. Of the 770 cases which showed shortening of the lower extremity, 395, or 51.30 per cent, had shortening of 5 mm. or less, while the remaining 375, or 48.70 per cent, had amounts of shortening which exceeded 5 mm.

As can be readily realized, whenever there exists a difference in the height of the femoral heads with apparent normal hip joints, that is, ruling out such conditions as dislocation, wandering acetabulum, and the like, there must necessarily be a tilt of the pelvis. As can be readily determined by measuring the height of the iliac crests, this tilt corresponds approximately to the difference in the height of the femoral heads. We have consistently observed ever since roentgenograms have been made in the upright position with the use of the stabilization device, that whenever there is such a pelvic tilt, there exists coincidentally a scoliosis of the lumbar spine (see Fig. 2 *B*). Because this scoliosis, in all instances, compensates for the tilt of the pelvis, it is referred to by us as compensatory scoliosis. The existence of this compensatory scoliosis in the presence of a tilted pelvis due to shortening of one or the other lower extremity is believed by us to have clinical significance and, furthermore, it is our opinion that the existence of any such condition cannot be determined with any degree of accuracy on gross physical examination. Furthermore, it becomes immediately apparent that the making of roentgenograms of the lumbosacral spine in the recumbent position, as is frequently done, completely prevents the discovery of such pathology as this.

In reviewing the 1,000 cases for roentgenological pathology, both the anteroposterior and the lateral views were carefully examined and all types of pathology were recorded. Because oblique views were not used, arthritic change in the apophyseal joints was not determined. Table II presents the various types of roentgenological pathology found and, as is apparent, the types have been classified on the basis of whether the patient had lower extremities of equal or unequal length. Accordingly, since 63 patients of the 230 with extremities of equal length had a pathological condition, this represents 27.38 per cent. On the other hand, the 230 cases of roentgenological pathology in those patients who showed a difference in leg lengths, represents 29.74 per cent. This makes a difference of 2.36 per cent between the two groups showing pathology and because of this close approximation, over 1,000 cases, one may assume that the existence of a difference in the lower extremity length has no correlation with the existence of roentgenological pathology. Inasmuch as the ratio between cases of equal lower extremities and of unequal lower extremities is approximately one to three, it can readily be seen that this ratio is fairly consistent in any one type of pathology listed. For example, if one were to refer to cases of spondylolisthesis, the ratio is seven to twenty for those of equal lower extremity lengths as of those of unequal lower extremity lengths which is approximately one to three. It will be noted that among the type of pathological conditions listed, there is a classification "structural scoliosis." It is felt that this deserves an additional word. Whenever a scoliosis was seen on the anteroposterior view which

TABLE I

MEASUREMENTS OF LOWER EXTREMITY LENGTHS
(1000 cases)

Lower Extremity Lengths	Millimeter Difference	Total Millimeter Difference	Number of Cases	Average Millimeter Difference
Equal	None	None	230	None
Right shorter than left	0– 5	665	199	3.34
	6–10	963	119	8.09
	11–20	1128	78	14.47
	21 over	278	10	27.80
TOTAL		3034	406	7.47
Left shorter than right	0– 5	604	196	3.08
	6–10	836	106	7.88
	11–20	739	55	13.43
	21 over	188	7	26.71
TOTAL		2367	364	6.50
TOTAL CASES			1000	

TABLE II

PATHOLOGICAL CONDITIONS SEEN ON ROENTGENOGRAMS

Pathology on Roentgenogram			Equal Lower Extremities	Unequal Lower Extremities			Total
				Right Shorter	Left Shorter	Total	
Sacroiliac joint arthritis	Right joint		1	4	3	7	8
	Left joint		7	6	6	12	19
	Bilateral		6	15	7	22	28
Existence of an anomalous joint between the transverse process of L-5 and sacrum	No arthritis	Rt. side	5	3	3	6	11
		Lt. side	3	8	7	15	18
		Bilateral	1	1	1	2	3
	With arthritis	Rt. side	0	2	2	4	4
		Lt. side	4	4	2	6	10
		Bilateral	0	1	0	1	1
Fusion between transverse process of L-5 and sacrum	Right side		0	0	2	2	2
	Left side		1	1	1	2	3
Spondylolisthetic lesions	Prespondylolisthesis		1	3	6	9	10
	Spondylolisthesis		7	8	12	20	27
	Reverse spondylolisthesis		2	3	1	4	6
Flat back (loss of normal lordosis)			7	13	5	18	25
Increased lumbosacral angle (above 50°) (elevated sacrum)			3	21	19	40	43
Asymmetry of articular facets of L-5			4	4	7	11	15
Structural scoliosis			6	4	4	8	14
Narrowed intervertebral disc space			1	7	2	9	10
Old compression fracture			2	1	4	5	7
Biconcave discs			0	9	6	15	15
Spina bifida (marked)			1	3	1	4	5
Osteoarthritis of the lumbar vertebra			0	2	1	3	3
Schmorl's nodes			1	1	1	2	3
Hemangioma of the lumbar vertebra			0	0	2	2	2
Anomalous joint between left transverse processes of L-2 and L-3			0	1	0	1	1
TOTAL			63	125	105	230	293
Complete Number of Cases			230	406	364	770	1000
Percentage with Pathology			27.38	30.78	28.84	29.74	29.30

existed and was not compensatory to any pelvic tilt and was due to another etiological factor, it was recorded as a structural scoliosis. Lacking in the table are the number of cases of compensatory scoliosis. As was pointed out previously, this condition existed whenever there was a difference in leg lengths. Furthermore, while no measurements were made to be presented in this particular study, it was a general consistent observation that the degree of scoliosis was proportionate to the degree of pelvic tilt. This, of course, becomes an evident fact with the realization that an individual who has a shortened leg will have to compensate completely if he intends to hold the upper portion of his body erect or in the midsagittal plane. The 8 cases of structural

scoliosis listed in those instances in which there was a shortened lower extremity were apparent and were due in all instances to associated pathology such as bony change due to old trauma involving either the vertebral body, the articular facets, or the intervertebral disc. In these 8 cases the patient did not have a compensatory scoliosis and it was apparent on examining the patient physically that there was an abnormal gross curvature of the spine.

A further analysis and comparison of the tabulation of the lower extremity mensuration and roentgenological pathology manifests a factor which the authors believe to be significant. When the group manifesting a difference in lower extremity lengths in excess of 5 mm. are studied from the standpoint of pathology, it is found that of this group of 375 patients, only 93, or 24.80 per cent, give evidence of roentgenological pathology while the remaining 282, or 75.20 per cent, show no evidence of roentgenological pathology other than the leg shortening with the corresponding tilt of the pelvis and the compensatory scoliosis

TABLE III

CORRELATION OF PATHOLOGY AND LOWER EXTREMITY SHORTENING IN EXCESS OF 5 MILLIMETERS

Cases with Lower Extremity Shortening in Excess of 5 mm.	Number	Per Cent
With roentgenological pathology	93	24.80
Without roentgenological pathology	282	75.20
TOTAL	375	100.00

of the lumbar spine (see Table III). With the realization, as stated in the beginning of this paper, that every soldier has been examined from the roentgenological standpoint because he had a low back complaint, it is evident that marked differences, that is a difference exceeding 5 mm. between lower extremity lengths is apparently associated with a low back complaint or disability. In other words, of every 4 soldiers who have a low back complaint and who show an inequality in lower extremity length in excess of 5 mm., only 1 out of the 4 shows roentgenological change as listed

TABLE IV

MEASUREMENTS OF LOWER EXTREMITY LENGTHS (100 Cases)

Lower Extremity Lengths	Millimeter Difference	Total Millimeter Difference	Number of Cases	Average Millimeter Difference
Equal	None	None	29	None
Right shorter than left	0– 5	74	19	3.89
	6–10	151	19	6.10
	11–20	35	3	11.66
	21 over	0	0	0
TOTAL		260	41	6.34
Left shorter than right	0– 5	56	19	2.94
	6–10	74	10	6.80
	11–20	11	1	11.00
	21 over	0	0	0
TOTAL		141	30	4.70
TOTAL CASES			100	

in Table II which might account for the soldier's symptoms, or the remaining 3 show no such roentgenological change and only manifest shortening of leg with corresponding tilt of pelvis and the compensatory scoliosis of the lumbar spine. For this reason, it is our opinion that the existence of such a condition is significant from the standpoint of symptomatology and disability.

As stated at the beginning of this article, every one of these soldiers was examined because of a low back complaint. In order to make some comparison, a group of 100 general duty soldiers who gave no evidence of physical complaint was selected by company commanders and examined in a similar fashion. Mensuration of lower extremity lengths reveal that within this group 29, or 29 per cent, had legs of equal length as shown in Table IV. Of those

having a shortened lower extremity, 41 cases had shortened right lower extremities and 30 cases had shortened left lower extremities. As seen in Table IV the average millimeter difference for the shortened right lower extremity is 6.34 mm., and 4.70 mm. for the shortened left lower extremity. These averages are definitely less than those found for the 1,000 soldiers with back complaints (Table I).

Analyzing these 100 cases from the standpoint of pathology seen on the roentgenograms, it was found that only 8, or 8 per cent revealed any evidence of roentgenological pathology. As may be recalled (Table II), 293, or 29.30 per cent, had evidence of roentgenological pathology in the series of 1,000 cases with back complaint. Of the 8 cases with pathology seen, 4 were cases of structural scoliosis. The remaining 4 were cases in which an anomalous joint had formed between the transverse process of the fifth lumbar vertebra and the wing of the sacrum, and 1 of these 4 cases revealed evidence of arthritis within the anomalous joint. Further analysis of the 100 general duty soldiers reveals that there are 33 cases in which the difference in lower extremity length exceeds 5 mm. Of these only 1 showed any evidence of roentgenological pathology. It will be recalled (Table III) that a similar analysis in the group of 1,000 revealed that of the 375 cases with lower extremity shortening in excess of 5 mm., 93 had evidence of roentgenological pathology.

The observations presented here are not made for the purpose of drawing absolute conclusions. These are assumptions made by the authors based on a technique of roentgenography which we believe to be unique and has great practical value, and only after further additional careful studies and observations are made by the use of such, or similar technique, may any positive conclusions be drawn.

SUMMARY

Observations have been presented on a series of 1,000 lumbosacral spines examined in the X-Ray Department of the Regional Station Hospital of Fort Leonard Wood, Missouri. The technique of examination is described and it is unique in that it utilizes a new stabilization device which necessitates that all lumbosacral spines be examined in the upright position. By this method it is possible to accurately measure differences in lower extremity lengths as manifested by a difference in the heights of the femoral heads.

The 1,000 cases were reviewed, both for the purpose of measuring difference in lower extremity lengths, and also from the standpoint of roentgenological pathology. Of the 1,000 cases 230, or 23 per cent, were found to have legs of equal length while the remaining 770, or 77 per cent, had lower extremities of unequal length. The average shortening of the right lower extremity was 7.47 mm. while the shortening of the left lower extremity was 6.50 mm.

The cases were reviewed for roentgenological pathology and it was found that of the 1,000 cases, 293, or 29.30 per cent had evidence of roentgenological pathology. Of this number, 63, or 27.38 per cent, were of the 230 cases of equal leg lengths, while 230, or 29.74 per cent were of the remaining 770 cases of unequal leg lengths.

A consistent observation which has been made is that in those cases with a shortened leg there is a corresponding tilt of the pelvis and a compensatory scoliosis of the lumbar spine. While the existence of roentgenological pathology does not seem to be correlated in any way with difference of leg lengths, it was found that in most cases with a difference in leg lengths in excess of 5 mm. and there were 375 such cases, only 93, or 24.80 per cent, had any roentgenological pathology while the remaining 282, or 75.20 per cent, had no roentgenological pathology other than the shortened leg with the corresponding tilt of the pelvis and the compensatory scoliosis of the lumbar spine. Since all of these had low back complaint it is possible that the remaining 75.20 per cent had complaint in association with the changes seen due to a shortened extremity.

A similar analysis was done on 100 general duty soldiers who were selected by their company commanders as individuals who had no physical complaints. The analysis showed that a slightly greater percentage had legs of equal length and the average amount of difference of lower extremity lengths was definitely less than in the series of 1,000 cases with low back complaint. Furthermore, the amount showing any evidence of roentgenological pathology was small, being 8, or 8 per cent, as compared with 293, or 29.30 per cent, for the series of 1,000 cases. Finally, among the 100 general duty soldiers with lower extremity differences in excess of 5 mm. only one showed any evidence of roentgenological pathology.

It is not our intention to draw any absolute conclusions from this study, but simply to present this technique which we believe manifests changes which are not brought out by other roentgenographic technique. Before absolute conclusions can be drawn, further observations, studies and analyses must be made.

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LARGE SUBLINGUAL METALLIC FOREIGN BODY

REPORT OF A CASE

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IN WARTIME it is natural that many cases with unusual findings will pass through the various departments of a hospital, and where trauma is the cause for complaint, the roentgen departments see all of them as a rule. We recently had a most unusual case in our Roentgen Department, and its findings were of extreme interest to us.

The patient was an enlisted man, aged twenty-one, gunner on a heavy bomber. While his plane was over the enemy target, an anti-aircraft shell burst near it; several fragments penetrated the plastic turret top where the patient was sitting, and one of these struck him in the mouth. His lips were cut, and several teeth were knocked loose. His tongue felt as though it was cut, and there was bulging under its left side, causing difficulty in expectorating the blood which accumulated, and in talking. He was unable to close his mouth completely. There was little pain. His mates on the bomber gave him first aid consisting of morphine, sulfanilimide crystals locally to the lips, a bandage to the mouth, and oxygen from the plane's supply.

On return from the mission to this base some four hours later, he was admitted to this hospital. Examination made in the Receiving Office showed the lower lip to have a deep cut in its mid-portion, and the upper to be nicked. There was a cut in the tongue for approximately 1 cm. of its length in the anterior portion on the left. Three of the front teeth were missing from their sockets—the upper right central incisor, and both lower central incisors. Two of these teeth were found lying loose anterior to the tongue and were lifted out. Several of the roots of these teeth were fractured and were seen still in their sockets, but loose. The patient was unable to completely close the mouth. He was having difficulty in expectorating the mixture of blood and sputum which accumulated.

Roentgenograms were made of the mandible and mouth region in posteroanterior, right and left lateral positions. An additional roentgenogram was made in the verticosubmental position, and occlusal films of the anterior teeth were exposed. These roentgenograms showed a very large metallic foreign body lying in a more or less horizontal plane in the left sublingual region of the mouth; its long diameter was directed in an almost true anteroposterior direction. By actual measurement following removal this foreign body was 5.3 cm. in length and 3 cm. in

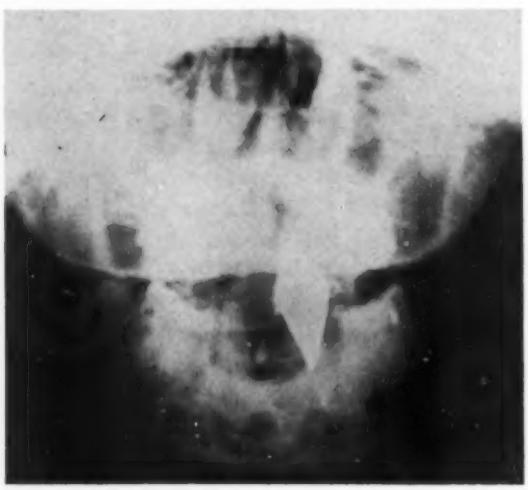

FIG. 1. Posteroanterior view of mandible showing large foreign body. The anterior portion of the mandible shows a concave fracture line at the symphysis, and several anterior teeth missing.

FIG. 2. Left and right lateral views of mouth, showing the jagged nature of the foreign body. Its size is best seen on these views.

width. Its edges were jagged. Several other opaque shadows, small in size, and seen best on lateral view, appeared to be fillings in teeth. The occlusal roentgenograms showed several of the front teeth missing, but a few of their roots remaining, as had been seen on physical examination. There was a small area of bone scooped out of the upper border of the mandible in its anterior portion. There were no gross fractures of the mandible or of the maxilla.

The patient was taken to the operating room, and under local anesthesia, the large foreign body was removed. There was no difficulty experienced by the surgeon in removing it, although it lay deep in the floor of the mouth and was entirely covered by the soft tissues. Several small bits of bone were attached to the metallic body, and in addition, several tooth fragments came out of the sublingual region on irrigation. The remaining tooth roots were extracted. Sutures were taken in the lower lip and in the upper gum. The sublingual wound was not closed.

Following operation, the patient was given mouth washes, and during the ten days he was observed here prior to his removal to a rear area hospital, his course was uneventful.

Two findings in this case were of especial interest to us. One was the unusual size of the metallic foreign body, particularly when compared to the relative size of the body part in which it was lodged. The other was the minimal amount of trauma done to the soft tissues, teeth and mandible when the foreign body entered the mouth. Our conception of this finding was that the patient must have had his mouth open at the time the fragment penetrated, a condition which he cannot recall due to the heat of battle at the moment, and that the foreign body must have entered in about the same plane in which it was found, which would present its smallest diameter to the oral aperture. Otherwise, we believe that the soft tissues and mandible would have sustained far greater trauma than they did.

Fig. 3. Verticosubmental view. The foreign body lies to the left of the midline.

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THE ROENTGEN TREATMENT OF BURSITIS OF THE SHOULDER*

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ROENTGEN therapy has been used for bursitis of the shoulder for a sufficiently long time to establish it as one of several recognized forms of treatment. Many papers, especially those by Lattman,[10] de Lorimier,[5] Sandström,[12] Herrman,[7] Jones,[8] Pendergrass and Hodes,[11] Baird,[1] Klein and Klemes,[9] Chapman,[4] Harris,[6] Brewer and Zink,[2] and Borak,[3] stress the results from roentgen therapy, so valuable information concerning its therapeutic effects is available in the literature.

This paper is based on the results obtained in 87 patients with bursitis of the shoulder, treated in the Roentgen Therapy Department of the Temple University Hospital, principally during the past six years. More than this number received treatment during this time, but only those with complete follow-up records were selected for this study. In order to be certain that the data concerning the effect of the treatment are reliable, the patients were followed for many months in the out-patient department. Although the observation period was unnecessarily long in many cases, it afforded an opportunity to obtain valuable information about recurrences and allowed the examining physician to check previous statements of the patient in regard to the effect of the radiation.†

TABLE I

BURSITIS—AGE INCIDENCE

25–29	30–39	40–49	50–59	60–69	70–79	80+	Total
Acute							
1	6	1	4	4			16
Subacute							
2		8	6	6	1		23
Chronic							
3	7	13	18	6		1	48
Grand Total							
6	13	22	28	16	1	1	87

The cases were placed in one of three categories, depending upon the duration of the symptoms. Those with symptoms for one week or less were classified under the heading, acute bursitis. The designation, subacute, applies to patients with pain and shoulder disability lasting more than a week and as long as two months. When the complaints were of longer duration than two months, they were considered as chronic in nature.

Table 1 shows the incidence of the three forms of bursitis in the various age periods. In each of the three groups, females predominate. In fact, in the group with chronic disease, the ratio is two to one. Fifty-six of the total of 87 patients are females.

Before the institution of roentgen treatment, a real effort was made to establish a diagnosis by obtaining a careful history and evaluating it with the clinical and roentgen findings. The clinical findings in the acute form are characterized by agonizing pain in the shoulder, aggravated by the slightest motion of the arm. In the three types, the pain is usually worse at night and becomes excruciating when the patient inadvertently rolls on the affected shoulder while in bed. In the usual so-called subdeltoid form, the discomfort is particularly marked over the greater tubercle and it may radiate in all directions from this area. Point tenderness is elicited in the greater

† Dr. W. Edward Chamberlain and I are indebted to our Consultant in Oncology, Dr. John V. Blady, for the organization of the follow-up clinics which has made it possible for me to report these cases.

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tubercle region and there may be evidence of local heat and swelling. The motion of the arm is greatly limited and attempts to rotate or abduct it cause the patient severe pain. Less severe pain but considerable restriction in motion is found in the subacute and chronic forms.

In the subacute and chronic forms, the diagnosis is often more difficult. It is important to obtain as much information as possible about the general condition of the patient because chronic shoulder pain is often on the basis of disease other than bursitis. Recent and old fracture, arthritis of the cervical spine and shoulder, brachial neuritis, coronary artery disease with angina, occupational strain and primary and metastatic bone disease are some of the conditions that produce shoulder pain simulating bursitis. Radiation therapy produces a mildly favorable transitory effect or no benefit at all in a painful shoulder due to any one of the above-mentioned abnormalities. Although little is expected from the use of radiation when the shoulder pain is on the basis of one of the above-mentioned conditions, treatment is usually inevitable once the diagnosis of bursitis and the possible benefit of roentgen therapy is suggested to the patient.

Roentgen examination of the shoulder is a necessary preliminary procedure, especially in the chronic type, to rule out or establish the presence of arthritis, old and recent fracture, osteoporosis, primary and metastatic bone disease, and calcification in the soft tissues adjacent to the head of the humerus.

In the past, considerable emphasis has been placed on the importance of calcification in the soft tissues in the diagnosis of bursitis of the shoulder, yet, experience indicates that the history and clinical findings are much more reliable in the evaluation of active disease than the demonstration of calcium deposits by roentgen examination. Interpreted in a strict sense, the presence of calcium deposits indicates previous inflammatory or degenerative disease in the tendon or bursa, but is unreliable evidence of active disease since in many patients the calcification is present without symptoms while others with severe symptoms are negative for calcifications. Experience in the past indicates that favorable results with irradiation are not influenced by the presence of calcification and are not dependent on the disappearance of calcium deposits, so no attempt was made to determine the length of stay of the calcium in the tissues after treatment.

The usual site of the calcium deposits is adjacent to the greater tubercle of the humerus in the tendon of the supraspinatus muscle, but occasionally they are found elsewhere in the soft tissues in the neighborhood of the head of the humerus. Supplementing the usual stereoscopic study of the shoulder with anteroposterior roentgenograms, exposed with the arm internally and externally rotated, increases the incidence of positive findings. The incidence of calcification in the affected shoulder in this series of 87 patients is 43.6 per cent. The percentage of patients with calcium deposits in the affected shoulder is practically the same in the three groups, there being a variation of only 0.4 per cent, as revealed by Tables II, III and IV.

The physical factors employed in the treatment are 180 kv. (av.) constant potential, 8 ma., 0.5 mm. of copper plus 1 mm. of aluminum filter and 50 cm. target-skin distance. With few exceptions, the individual dose was 150 r, measured in air, at the skin without backscatter, delivered through one field either 10 by 15 or 15 by 15 cm. in size. The beam was either directed to the anterior or anterolateral aspect of the shoulder, depending upon the size or site of the involved area.

Most of the acute cases received from one to three daily doses with an additional dose in one week if necessary. Several in this group of 16 were completely relieved by a single dose and a few needed only two treatments. Many of the 23 patients with subacute manifestations were treated twice a week for two weeks for a total of four treatments. A few received 150 r daily for

TABLE II

ACUTE BURSITIS

Pain 1 week or less before treatment

Complete relief—14	10—Relief in week or less (5 of these well in 3 days or less) 4—Relief in 10–14 days—Well in 3 weeks or less
Poor result—2	1—Prompt relief but pain recurred in 3 weeks 1—Slight relief only
Treated—16	

Calcification in 7 of 16 or 43.7 per cent

three days and a fourth treatment in one week.

Forty-eight patients with chronic shoulder pain were treated in one of three ways, depending upon the severity of the symptoms and the choice of methods by the radiologist administering the therapy. Twenty-nine were treated twice a week for two weeks, 11 received weekly doses usually for four weeks, and in 8, doses were administered on three consecutive days with a fourth treatment in one week's time. As far as it is possible to determine, the interval between treatments made no difference in the results in the patients with chronic disease.

The results in 14 of the 16 patients with acute symptoms, one week or less, were excellent, as indicated by Table II. Ten of the 14 were completely relieved of pain in one week and of these, 5 were well in three days. The additional 4 reported a cure in three weeks or less, with marked relief of symptoms from ten days to two weeks. The 2 remaining cases did not respond well to treatment. One obtained a prompt response but the pain recurred in three weeks, while the other reported some alleviation of the acute symptoms but persistence of mild pain for many days following the treatment.

The responses in 19 of the 23 patients with subacute symptoms were good but not as prompt as seen in the acute cases (see Table III). Six were well in one week or less and the majority of the remaining 13 reported considerable relief of the pain after treatment, with persistence of mild symptoms for about two weeks. In 2 of the 13, symptoms lasted for as long as two months. Four patients in the subacute group failed to respond satisfactorily to treatment (Table III).

Roentgen therapy yields relatively poor results in chronic bursitis, as shown by Table IV. In 16 of 48 cases, or 33⅓ per cent, a good result was obtained. When favorable, the response was slower than that noted in acute and subacute bursitis, but most of those patients who were benefited reported relief and then disappearance of pain in from three to six weeks. A few were completely relieved in less than three weeks. In some, there was residual limitation of motion estimated as slight, possibly on the basis of adhesions or other inflammatory disease changes. Ten of the patients obtained a temporary relief but the pain recurred in from one to nine months. Usually, the pain relief lasted from two to six weeks, but in a few the result was of longer duration. Perhaps these results could be classified as partial relief, but, when the patients were interviewed months after the treatment, they considered the treatments ineffectual since the pain recurred. The remaining 22 were not benefited at all by the treatment. A careful

TABLE III

SUBACUTE BURSITIS

Pain 2 months or less before treatment

Complete relief—19	6—Well in 1 week or less 10—Prompt improvement but mild pain, 2 weeks 1—Pain relief in 3 weeks 2—Mild pain lasted 2 months
Poor result—4	1—Relief for 2 weeks, recurrence 1—Relief—3 mo., recurrence 1—Slight improvement? 1—No benefit
Total—23	

Calcification in 10 of 23 or 43.4 per cent

study of this group showed that in some of them the shoulder pain was on some other basis than bursitis. Since it was not feasible to defer the treatment long enough to adequately study the patients with atypical shoulder pain, they were treated and the negative results classed as failures.

TABLE IV

CHRONIC BURSITIS

Pain longer than two months before treatment.	
Complete relief—16	4—Relief 1 month or less 3—Relief 6 weeks or less 9—Exact duration of pain after treatment not known; follow-up indicated complete relief
Poor result—10	9—Relief only 2 months 1—Relief only 9 months
No result—22	No relief from pain
Total—48	
Calcification in 21 of 48 or 43.7 per cent	

To determine whether the responses differed in those patients classified as acute and subacute whose history was negative for previous attacks of bursitis from those with a positive history for previous episodes, another grouping was made, termed recurrent. Only 9 patients make up this group. All were pain free from time of the previous attack of bursitis that occurred a number of months or several years ago until the present one. Some of this group sought roentgen therapy because they previously obtained relief from its use, while others recovered spontaneously from a previous attack or were treated by other methods. With one exception, the responses in this group were good so the results are comparable to those obtained in patients who never experienced previous attacks. The results are included with the conventional acute and subacute cases so the recurrent group is not a separate one as far as the statistics are concerned.

SUMMARY AND CONCLUSIONS

The results obtained with roentgen therapy in eighty-seven patients treated for bursitis of the shoulder indicate that it is a valuable form of treatment for those cases with acute and subacute manifestations of the disease. All but two of the patients with acute bursitis were relieved of pain, and the majority were well in one week or less. The responses in the subacute group were not as prompt, but more than two-thirds reported complete relief of pain in two weeks or less and only a few were not benefited. Only one-third of the group with chronic symptoms were cured, and almost 50 per cent reported no relief of pain.

The relatively poor results with chronic bursitis may be due in part either to adhesions or inflammatory soft tissue changes and to a lesser degree to an erroneous initial diagnosis. The numerous causes for chonic shoulder pain simulating bursitis, and the lack of response of most of these conditions to roentgen therapy, emphasize the necessity for careful clinical and roentgen examinations before the decision is reached to administer treatment.

The presence of calcium in the soft tissues of the shoulder is of lesser importance than the history and clinical findings in the diagnosis of bursitis. The incidence of calcification in the affected shoulder of the patients in this series is 43.6 per cent. Since favorable results with roentgen therapy are not dependent on the disappearance of calcium deposits, no attempt was made to determine their length of stay in the tissues after treatment.

The results with recurrent bursitis are comparable to those obtained in patients with an initial attack, so irradiation should not be withheld from a patient with acute or subacute bursitis simply because the history indicates an episode of the disease at some time in the past. However, the lack of response when dealing with chronic bursitis is sufficient reason to withhold further irradiation as none of the patients in this group benefited from a second series when

the results from the first series were indeterminate or poor.

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IRRADIATION SICKNESS IN RATS*

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INTRODUCTION

IT IS well known that the welfare of many persons with tumors, leukemia, and various types of dermatitis is dependent upon the administration of sufficient roentgen-ray dosage to control the condition. But severe irradiation sickness often makes it difficult to give adequate therapy. The action of radiation on living matter is of such complexity that treatment for the prevention of irradiation sickness has remained of necessity on a nonspecific basis. There is no known mechanism by which the physician can be certain of protecting every atom and molecule of body tissue. The living tissue protoplasm is a complex material consisting of water, proteins, lipoids, carbohydrates, electrolytes, enzymes, hormones, and perhaps many other organic compounds. Each cell of the human body is an exceedingly complicated unit of physico-chemical equilibrium. During irradiation manifold chemical changes are produced in regions where the energy quanta are absorbed. With large dosage, disorganization of the cell constituents results and significant effects, both beneficial and detrimental to the patients, are produced. These radiobiologic reactions are largely determined by the size of the dose, the quality of the wavelength of radiation, and the frequency and manner of its application.

In the Nutrition Clinic in Birmingham, Alabama, the problem of irradiation sickness has been one of great interest for seven years. Our studies[1,2] have made the use of careful dietary and vitamin therapy seem logical, if on empiric grounds alone, since nutrition is often deranged in persons needing radiotherapy. Accordingly, it was desired to determine more specifically the importance of the nutritional level and the value of various dietary and parenteral supplements. Rats were chosen for the study of irradiation sickness reported in this communication since they so closely resemble human beings in many nutritional and metabolic characteristics and since they enable more adequately controlled experiments.

MATERIALS AND METHODS

For this study, which has been carried out over a period of eighteen months, 824 Sprague-Dawley albino weanling rats were used in the twenty experiments, usually in groups of fifty-two. The experiments were begun when the rats were twenty-six to twenty-nine days old. They were continued until the rats were from fifty-four to ninety-five days old. The rats were housed in individual cages, and daily individual weight records were kept. Each rat was provided with the prescribed diet and with water *ad libitum*. Adequate control groups which were not exposed to roentgen radiation were used to parallel fully the test conditions in each experiment.

The test rats were given a single massive dose of radiotherapy varying from 600 roentgens to 800 roentgens in the different experiments. The factors used were 200 kv. or 210 kv., 15 ma. or 20 ma., 50 cm. distance, 0.5 copper and 1 mm. aluminum filter, and a 20 by 20 cm. cone. For this exposure the rats were arranged in wooden boxes 20 by 20 cm. which were divided into

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Grateful acknowledgment is also made to the Cardiac Laboratory and to Clarence A. Mills, M.D., University of Cincinnati, for granting us space in their laboratories for housing some of the animals.

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eight equal compartments. One rat was placed in each compartment and the box was covered with a wire screen. The compartments were of such a size that the rats were able to assume any elongated position; hence it is thought that the total irradiation was fairly uniform over the entire surface of the body. The roentgen dosage was measured by means of an integron placed directly above the box containing the rats. It was necessary to determine the critical dosage level for each set of experimental conditions so that approximately one-half of the rats would die and the other half would survive the irradiation. At the time of the roentgen treatment the age of the group of rats in the different experiments varied from thirty-eight days to ninety-one days. It was necessary to employ a massive dose of roentgen rays in order to obtain objective criteria for the incidence and severity of irradiation sickness. Those criteria used were percentage fatality, post roentgen therapy survival time, weight changes, observations on activity and food consumption.

In these experiments various diets were employed in order to establish different nutritional levels. The nutritionally complete diet consisted of Purina Dog Chow Checkers *ad libitum* (stock diet). In some cases this diet was limited in amount to 5 gm. daily, which was the quantity found experimentally to produce approximately balanced metabolism so that no appreciable weight changes occurred from day to day. Two essentially similar vitamin-free diets were also used in various experiments.

Vitamin-free Diet A

Cornstarch	68%
Vitamin-test casein	18%
Lard	10%
Salts (U.S.P. salt mixture II)	4%

Vitamin-free Diet B

Sucrose	68%
Vitamin-test casein	18%
Vegetable oil	10%
Salts (U.S.P. salt mixture II)	4%

The U.S.P. salt mixture II had the following composition:

Sodium chloride (U.S.P)	4.35%
Magnesium sulfate (U.S.P.)	13.70%
Sodium biphosphate (U.S.P.)	8.72%
Potassium phosphate (K_2HPO_4)	23.98%
Calcium biphosphate $CaH_4(PO_4)_2H_2O$	13.58%
Ferric citrate (U.S.P. reagent 17.5% Fe)	2.97%
Calcium lactate (U.S.P.)	32.70%

The value of daily oral vitamin supplements to the vitamin-free diet A was studied by administering them three days before the roentgen treatment and continuing daily thereafter until the termination of the experiment. The following daily supplements were dissolved in 5 cc. of water and added to each rat's usual vitamin-free diet:

Thiamine	0.2 mg.
Pyridoxine	0.2 mg.
Inositol	0.2 mg.
Riboflavin	0.2 mg.
Nicotinamide	2.5 mg.
Calcium pantothenate	10.0 mg.
Choline hydrochloride	20.0 mg.

Each rat was also given daily 0.5 cc. of a cod liver oil preparation which contained 850 U.S.P. units of vitamin A and 85 U.S.P. units of vitamin D per gram.

Parenteral medications which were tested were:

Vitamin B_6—in daily intraperitoneal doses of 0.5 mg. and 1.0 mg. dissolved in saline

Vitamin K—in daily intraperitoneal doses of 0.01 mg. and 0.1 mg. dissolved in saline

Thiamine and nicotinamide—in daily intraperitoneal doses of 0.5 mg. each dissolved in saline

Folic acid—in daily subcutaneous doses of 25 micrograms dissolved in saline and sodium bicarbonate solution.

Crude liver extract (Lilly, 1 U.S.P. unit per cc.)—in three subcutaneous doses of 0.2 cc., the first two days before the roentgen treatment, the second immediately after the roentgen treatment, and the third two days after the treatment.

With the exception of the crude liver extract, which was given in three single doses, all the parenteral medications were begun two days before the roentgen treatment and continued daily thereafter until the termination of the experiment. Control groups receiving saline injections were used to parallel each of the groups receiving any parenteral medication.

TABLE I

IRRADIATION SICKNESS IN RATS—RESULTS OF EXPERIMENTS XIX AND XX

Group	Number of Rats	Diet	Medication	Dosage (roentgens)	Experimental Days 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	Total Deaths	Per Cent Fatality
1	16	Vitamin-free	Saline	700										Daily medication begun		700 roentgens				X							X		X	X	X	X	X	X	X	X			16	100.00
2	16	Vitamin-free	Folic acid	700																							X	X	X	X	X	X	X	X	X	X	X	15	93.75
3	16	5 gm. stock	Saline	700																	X	X					X										X	X	5	31.25
4	16	5 gm. stock	Folic acid	700																						X													1	6.25
5	10	Vitamin-free	Saline	None																										X					X	X	X	9	90.00
6	10	Vitamin-free	Folic acid	None																												X	X	X			X	X	9	90.00
7	10	5 gm. stock	Saline	None																																			0	0.00
8	10	5 gm. stock	Folic acid	None																																			0	0.00

X = rat died.

RESULTS

Of the twenty experiments carried out, the results of the most significant ones will be reported. Experiments XI, XIX, and XX were of especial interest and are reported in detail in Figures 1–6 and in Table 1.

It was necessary to determine the critical roentgen-ray dosage level at which approximately one-half of the rats died within three weeks after the radiation exposure. Depending on the age of the rats, temperature and other experimental conditions, this dosage level was either 650 roentgens or 700 roentgens. Older rats were found to withstand the roentgen-ray exposure better than younger ones.

Forty-four and forty-six day old rats maintained on a vitamin-free diet were exposed to 650 r on the twentieth experimental day. They were given three doses of 0.2 cc. of Lilly crude liver extract (1 U.S.P. unit per cc.), the first dose two days before, the second dose the day of, and the third dose two days after the roentgen-ray exposure. These subcutaneous injections gave no indication of protective value against irradiation sickness.

Rats maintained on a stock diet were given intraperitoneal injections of 0.01 mg. or 0.1 mg. of vitamin K dissolved in 0.5 cc. of saline daily, beginning two days before the exposure to 700 r. These different groups of rats were 37, 42, 49, 56, and 60 days old at the time of the roentgen irradiation. This medication gave no significant evidence of prophylactic or therapeutic value in irradiation sickness. Intraperitoneal injections of 0.5 mg. or 1.0 mg. of pyridoxine (vitamin B_6) in other groups of rats under the same test conditions showed possible protective value. The evidence, however, was not statistically sufficient to warrant a definite statement.

Similarly, daily intraperitoneal doses of a combination of 0.5 mg. of thiamine and 0.5 mg. of nicotinamide dissolved in 0.2 cc. of saline gave no statistically significant evidence of protective value, though the results indicated such a trend. This medication was given to rats maintained on stock

diet and exposed to 700 r when thirty-eight days old.

Folic acid in daily subcutaneous doses of 25 micrograms dissolved in 0.2 cc. of saline and sodium bicarbonate solution also gave evidence of a trend towards possible protective value. This medication was tested on rats exposed to 700 r when thirty-eight days of age. The rats were maintained on a vitamin-free diet *ad libitum* or on the stock diet in an amount limited to 5 gm. This quantity of stock diet was found to maintain fairly balanced metabolism with minimal weight changes. Table 1 shows the results of the testing of folic acid in Experiments XIX and XX. These two experiments are reported together since they were in every way identical.

Experiments XIX and XX (Table 1) clearly demonstrate also the increased incidence of fatal irradiation sickness in rats maintained on a vitamin-free diet as compared with those maintained on a limited amount of stock diet (5 gm.). The importance of the nutritional status of a rat in determining the degree of irradiation sickness following exposure to roentgen radiation is also shown in Experiment XI. In addition, this experiment demonstrates the value of daily oral vitamin supplements of thiamine, pyridoxine, inositol, riboflavin, nicotinamide, calcium pantothenate, and choline hydrochloride, beginning three days prior to the roentgen-ray exposure. Previously these rats were maintained solely on a vitamin-free diet. On the twentieth experimental day, when these rats were forty-eight days old, they were given 650 r. Figures 1–6 show the weight curves and fatalities for each of the groups of rats in this experiment. The striking difference in the incidence of fatal irradiation sickness between those rats on

TABLE II

MEDICATIONS TESTED FOR PROTECTIVE VALUE AGAINST IRRADIATION SICKNESS IN RATS

Diet	Roentgen-Ray Exposure		Medication	Protective Value
	Age of Rat (in days)	Dosage (roentgens)		
Vitamin-free	44 or 46	650	Crude liver extract—three doses, 0.2 cc. subcutaneously	0
Stock	37, 42, 49, 56, or 60	700	Vitamin K—daily, 0.01 mg. or 0.1 mg. intraperitoneally	0
Stock	37, 42, 49, 56, or 60	700	Pyridoxine—daily, 0.5 mg. or 1.0 mg. intraperitoneally	+(?)
Stock	38	700	Thiamine and nicotinamide—daily, 0.5 mg. each intraperitoneally	+(?)
Vitamin-free	38	700	Folic acid—daily, 25 micrograms subcutaneously	+(?)
Vitamin-free	48	650	Daily oral supplements: Thiamine 0.2 mg. Pyridoxine 0.2 mg. Inositol 0.2 mg. Riboflavin 0.2 mg. Nicotinamide 2.5 mg. Calcium pantothenate 10.0 mg. Choline hydrochloride 20.0 mg.	+

the vitamin-free diet and those on the stock diet is clearly shown. In groups 2A and 2B, which are identical, the value of the oral vitamin supplements is demonstrated in that the number of fatalities is so markedly diminished. Table II presents a summary of the protective value in irradiation sickness of the medications tested.

In Experiment XI there was a considerable difference in size between the fats on the vitamin-free diet, and those on the unlimited stock diet. The rats on the stock diet weighed about twice as much as the deficient rats at the time of roentgen irradiation. It therefore seemed possible that the smaller rats received a greater and more penetrating dose of roentgen rays than did the larger rats. This then could partly account for the greatly increased number of fatalities in those groups. In the later experiments the amount of stock diet was limited to 5 gm. daily as in Experiments XIX and XX. With these controlled diets, all the rats were approximately the same size at the time of roentgen irradiation so that there can be no question of the fact that the dosage received by the rats of all the groups was the same. Since the results of all these experiments are corroborative, Experiment XI may still be considered significant in spite of the difference in size of the rats in different groups at the time of roentgen irradiation.

DISCUSSION

The difference in the incidence and severity of irradiation sickness in rats of different ages is of interest. It is a basic concept in radiotherapy that the younger and more rapidly growing cells are the most susceptible to the lethal effects of the rays. It is therefore interesting to find that the younger rats seem more susceptible to severe and fatal irradiation sickness than do older rats.

The weight curves of the rats in Experiment XI (Fig. 1–6) clearly show the usual pattern of weight changes following roentgen-ray exposure. Immediately after the roentgen irradiation, there is a primary

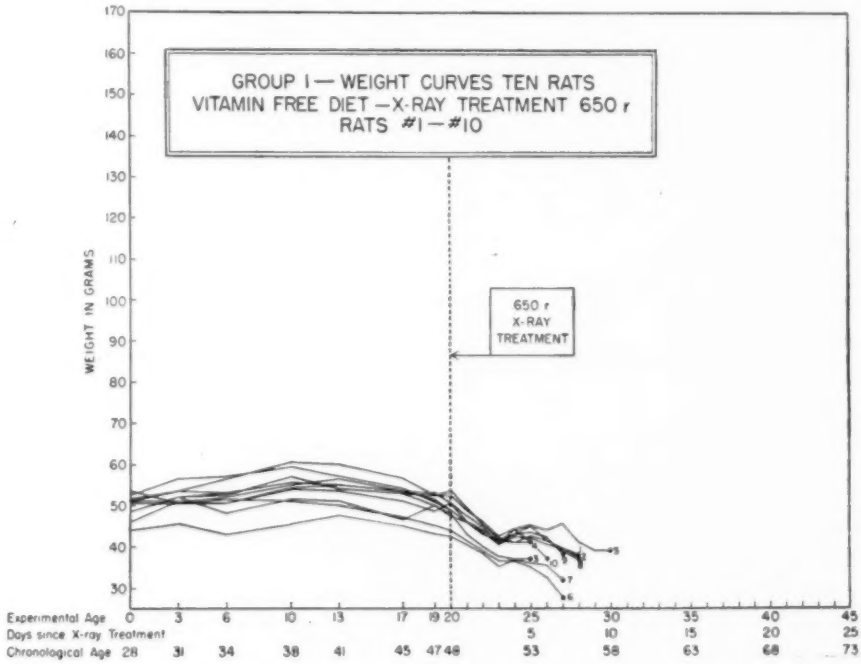

FIG. 1

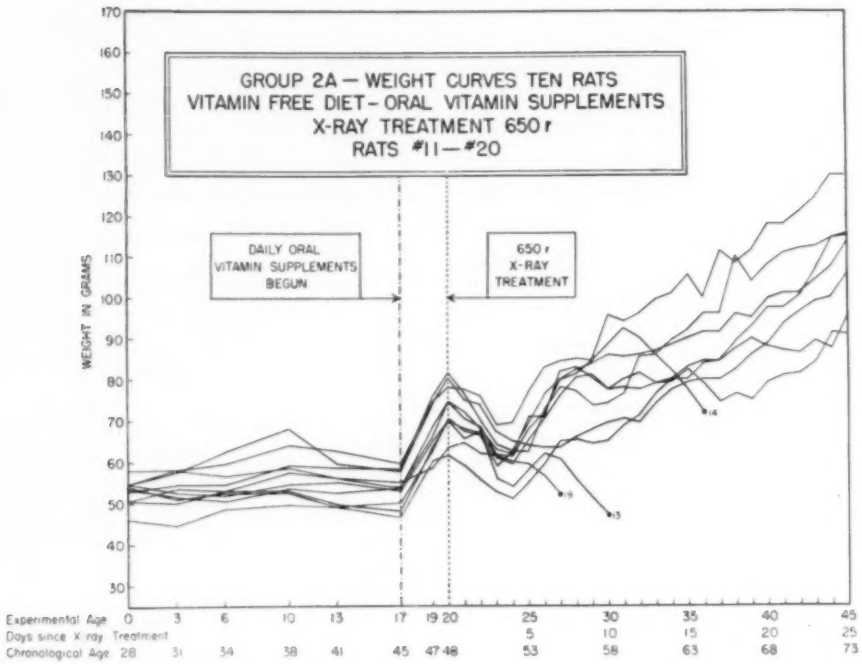
GROUP 2A — WEIGHT CURVES TEN RATS
VITAMIN FREE DIET - ORAL VITAMIN SUPPLEMENTS
X-RAY TREATMENT 650 r
RATS #11 — #20
DAILY ORAL VITAMIN SUPPLEMENTS BEGUN
650 r X-RAY TREATMENT
WEIGHT IN GRAMS
Experimental Age
Days since X ray Treatment
Chronological Age

FIG. 2

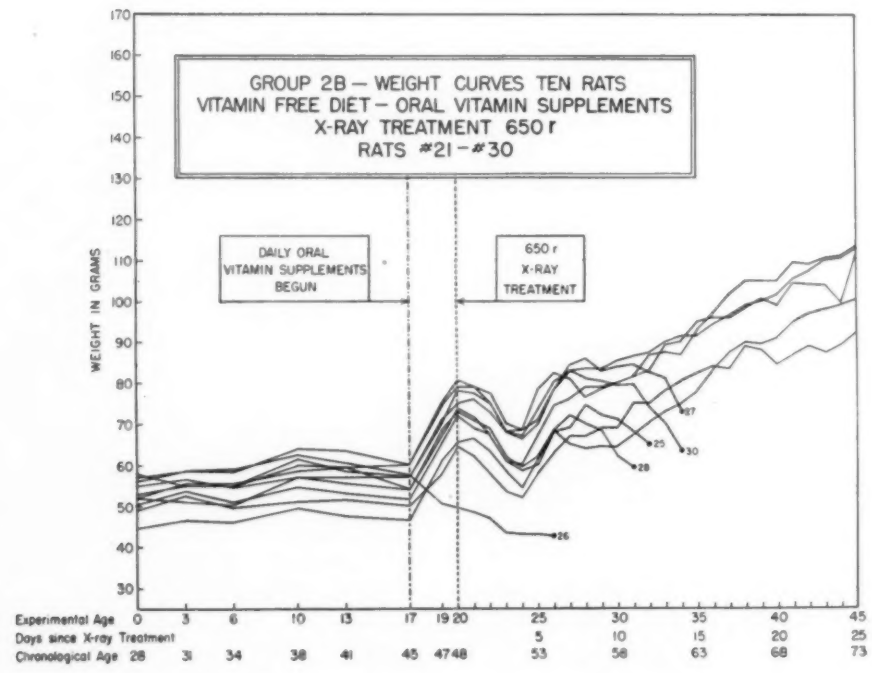
GROUP 2B — WEIGHT CURVES TEN RATS
VITAMIN FREE DIET — ORAL VITAMIN SUPPLEMENTS
X-RAY TREATMENT 650 r
RATS #21 — #30
DAILY ORAL VITAMIN SUPPLEMENTS BEGUN
650 r X-RAY TREATMENT
WEIGHT IN GRAMS
Experimental Age
Days since X-ray Treatment
Chronological Age

FIG. 3

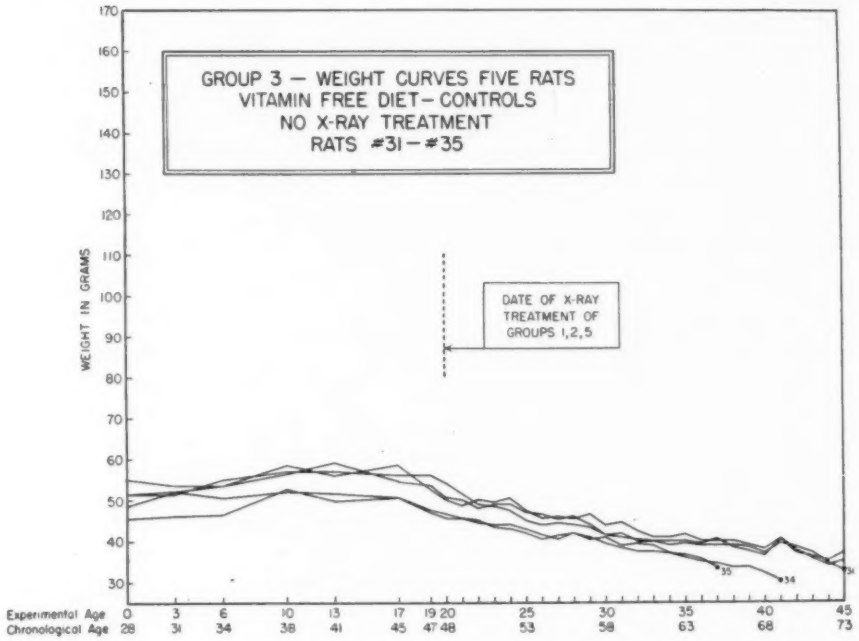
GROUP 3 — WEIGHT CURVES FIVE RATS
VITAMIN FREE DIET — CONTROLS
NO X-RAY TREATMENT
RATS #31 — #35
DATE OF X-RAY TREATMENT OF GROUPS 1,2,5
WEIGHT IN GRAMS
Experimental Age
Chronological Age

FIG. 4

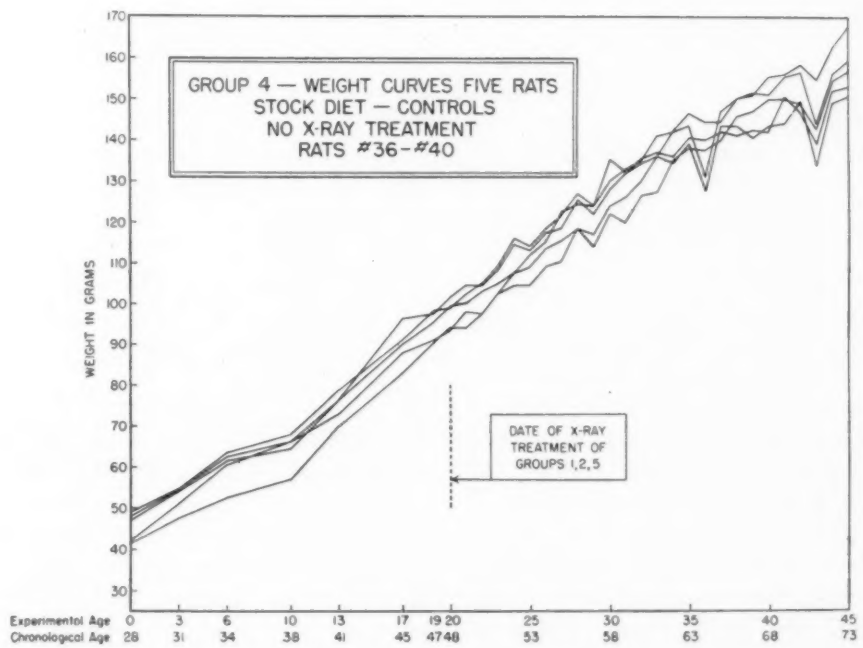
GROUP 4 — WEIGHT CURVES FIVE RATS
STOCK DIET — CONTROLS
NO X-RAY TREATMENT
RATS #36-#40
DATE OF X-RAY TREATMENT OF GROUPS 1,2,5
WEIGHT IN GRAMS
Experimental Age
Chronological Age

FIG. 5

weight-loss period of three to five days' duration, which may be terminated by death. This rapid weight loss is probably caused by a change in water metabolism. Subsequently the rats experience a period of weight gain which may last only a few days or which may continue indefinitely in pallor is always followed by death. All these facts can be used as criteria for the severity of the irradiation sickness in the rats.

It should be noted in Figure 4 and in Table I that many of the rats in the control groups maintained on the vitamin-free diets in Experiment XI and in Experiments

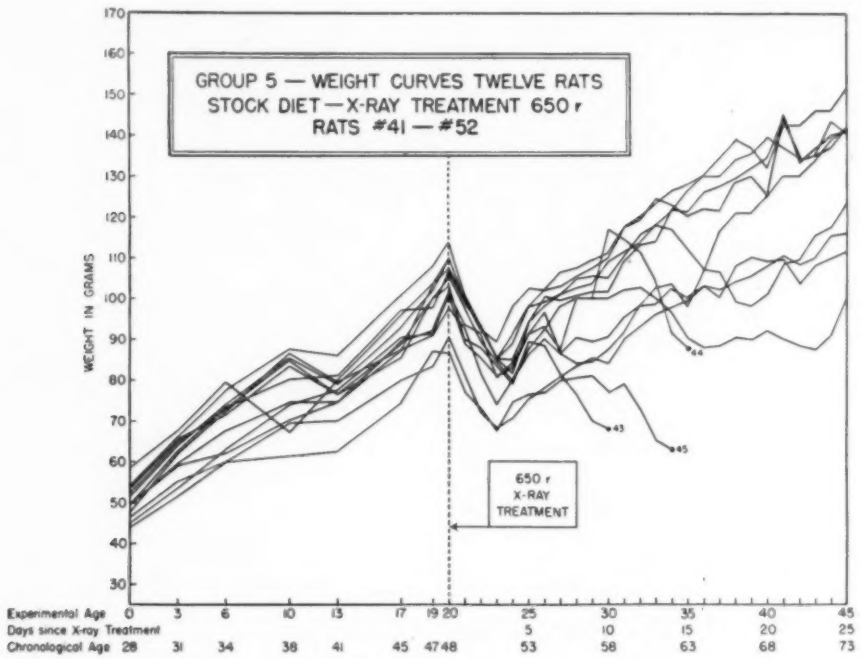

FIG. 6

the cases of the rats which fully survive the roentgen exposure. Some rats, however, then begin a second weight-loss period of varying length. If this occurs it is usually followed by death. Irregularities in growth curves may be caused by a temporary lack of water through experimental error.

Several general observations can be made of the rats after roentgen irradiation. The rats are noticeably less active, at least until the start of the weight gain period. The food consumption is markedly diminished. The fur becomes considerably loosened though it is not actually shed. There is often evidence of diarrhea. The rats may assume an extremely pale appearance so that the ears, eyes, feet, and tail become almost colorless. This severe XIX and XX also died, though they had not been exposed to roentgen radiation. This indicates the severity of the deficiencies which are produced by these vitamin-free diets. This factor must be recognized in the consideration of the fatalities following roentgen-ray exposure.

SUMMARY AND CONCLUSIONS

Criteria for the incidence and severity of irradiation sickness in rats following exposure to a single massive dose of radiotherapy were percentage fatality, weight changes, observations on activity and food consumption, and autopsy findings. The roentgen-ray dosage was determined to give approximately 50 per cent fatality under each set of experimental conditions.

These findings establish the fact that young rats on a vitamin-free diet showed marked increased susceptibility to fatal irradiation sickness when compared with rats maintained on an adequate diet. It is of considerable importance that we were able to demonstrate that the daily oral vitamin supplements of thiamine, pyridoxine, inositol, riboflavin, nicotinamide, calcium pantothenate and choline hydrochloride, begun three days before the roentgen-ray exposure, greatly decreased the incidence of fatal irradiation sickness in rats otherwise maintained on a vitamin-free diet. These observations on 824 rats under controlled conditions support our clinical observations in this Nutrition Clinic that persons with nutritive failure have increased susceptibility to irradiation sickness. Until the basic mechanisms of irradiation sickness are fully understood, treatment for its prevention and alleviation will necessarily remain on a nonspecific and symptomatic basis.

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UNUSUAL SKIN SENSITIVITY TO ROENTGEN RADIATION

REPORT OF CASE

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SKIN reactions in the course of ordinary diagnostic roentgenography are encountered but seldom under present day conditions although the possibility of such reaction must always be borne in mind and guarded against. The case to be presented here appears to be an unusually rare manifestation of skin sensitivity to roentgen radiation.*

A soldier, Negro, aged twenty-nine, reported to a battalion aid station on January 5, 1945,

FIG. 1. Left side (nearest the camera) showing well demarcated zone of "chocolate erythema" following spot lateral roentgenograms of the lumbosacral region.

because of painful external hemorrhoids. He was accordingly sent to an evacuation hospital and thence to a general hospital where the hemorrhoids were removed under local anaesthesia on January 18, 1945. As he complained of pain in the lower back and left buttock extending down the left leg, anteroposterior and lateral roentgenograms of the lumbosacral spine were made on January 21, 1945. No significant findings were noted. The patient was subsequently transferred to another general hospital and on February 18, 1945, the lumbosacral spine was again roentgenographed. He was finally evacuated to England and was admitted to this general hospital on February 25, 1945. He continued to complain of the same type of pain. His roentgenograms had either been left behind or lost in transit, so that on March 6, 1945, spot anteroposterior and right lateral views of the lumbosacral spine were made, two exposures being made in each position.

On March 15, 1945, his ward officer, Captain Weiss, noticed a perfectly round zone of "chocolate colored erythema" on the left side laterally over the crest of the ilium. This circle was found to correspond exactly to the size of the cone used in making the lateral exposure (Fig. 1). Then two similar but much fainter overlapping circles of erythema were discovered on the right above and below the crest of the ilium and another less well defined zone anteriorly in the suprapubic region. The patient stated that at the previous hospitals the lateral views were made with the right side towards the tube (whereas ours were made with the left side towards the tube) and the explanation became obvious. (All but the first circle were well demonstrated photographically and no infrared film was available.)

In order to demonstrate conclusively that these zones of "erythema" were produced by roentgen radiation on April 12, 1945, he was given a single exposure to the right mid-thigh laterally using 85 kv., 30 ma., for twelve seconds at a 30 inch tube-film distance—but actually 19 inch tube-skin distance. A cone $4\frac{1}{2}$ inches in diameter was in place down to 1 inch above the skin. The center of this area was marked with skin pencil. Ninety-two hours later a faint but definite round area of "chocolate erythema" appeared at this site. This zone became well demarcated and reached its maximum intensity on April 18, 1945, six days after exposure (Fig. 2). The intensity of coloration of this zone remained constant for about seven days and had faded but slightly two weeks later. The zone at the crest of the left ilium had diminished

* At least in the experience of the group of radiologists to whom this case was presented; no literature on this subject is available to the writer at this time.

definitely in intensity but was still quite distinct on May 6, 1945, two months after the exposure. The two overlapping zones on the right had faded appreciably and were losing their identity by May 6, 1945, a period of fifteen and seven weeks respectively after the exposure. The zone in the suprapubic region had also faded somewhat. By May 18, 1945, the fading had progressed definitely but slightly.

The original exposures were made with a 200 ma. Philips tube, oil immersed with an inherent filtration equivalent to at least 0.5 mm. Al but no added filtration.* The trial exposure to the right lateral thigh was made with a 100 ma. single focus, oil immersed Machlett tube, Aeromax "12," with an inherent filtration not over 0.5 mm. aluminum. It is not known what factors or types of equipment were used at the previous hospitals. With the same factors employed in making the trial exposure to the right thigh namely twelve seconds, 30 ma., 85 kv., at 19 inch tube-target distance with the same type of tube, Machlett Aeromax "12" (with a Westinghouse machine in place of the Philips originally used), it was found that 32 roentgens were delivered in air. The measurements were made with the Victoreen ionization chamber in Lieutenant Colonel Rude's laboratory. Subsequently in this department with a Philips 200 ma. tube and the Philips machine with the same factors mentioned above, it was determined that 23 roentgens were delivered in air. No sphere gap was available to check the voltages at the time of these two measurements but it is evident that different outputs are to be expected considering that different tubes, different types of transformers as well as slightly different line voltages were utilized. Still the anteroposterior views were made at approximately 24 inch tube-target distance, the exposure factors being 70 kv., four seconds at 30 ma., which even when multiplied by two would still be considerably less than that employed in the trial exposure.

It should be considered that the technique employed on this patient has been used many times on others apparently without untoward results. In fact, the customary procedure in studies of the lumbar and sacral spine has been to take anteroposterior and lateral views of the entire region and spot anteroposterior and lateral views of the lumbosacral region. In addition, oblique views have been made when indicated (occasionally stereoscopic). Since most of our patients were white, any erythema would have been noted very readily. To sum up all these factors, it seems evident this patient was unduly sensitive to roentgen rays.

Fig. 2. Right side (nearest the camera) showing zone of "chocolate erythema" ten days after trial exposure.

Fortunately, such sensitivity happens but rarely but since it does occur it serves to emphasize the importance of ascertaining what exposures have been made and the advisability of using additional filters. The importance to the patient of knowing this fact is obvious, and he has been cautioned to inform his physician regarding his sensitivity should he ever require further roentgen examination or more particularly roentgen treatment.

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* 1 mm. Al filter has since been secured for this tube.

A VISIT TO RÖNTGEN'S LABORATORY IN 1923

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COLONEL Etter's account* of his 1945 visit to the Physical Institute in war-ruined Würzburg recalls to my mind a visit that I made in the Spring of 1923, reported orally at the 1923 meeting of the American Roentgen Ray Society but until now have not published.

It was a rainy day in May, 1923, when I called at the University of Würzburg and presented my credentials to Professor von Frey, the head of the Physiological Institute. At that time I was a member of the faculty of the Peking Union Medical College and since my sabbatical year was financed in part by a fellowship from the Rockefeller Foundation I carried letters of introduction from some of the officers of that organization. Those letters opened von Frey's door for me despite the fact that he had to be called away from a class, but it

FIG. 1. Photograph of Röntgen at the age of sixty-three. Purchased from a professional photographer in Munich and presented to me in China by my friend, Dr. Adrian Taylor.

FIG. 2. Physical Institute as it appeared in 1923. The plaque which was attached to the building in 1905 freely translated reads: "In this building, in the year 1895, W. C. Röntgen discovered the rays that have been named for him." Colonel Etter's 1945 Figure 1*A* seems to be taken from about the same point. If this assumption is correct, then the years have seen several changes. The iron railing and vines have been removed. The corner window has been walled up and the old plaque has been replaced by lettering on the new masonry at the former window site.

* Etter, L. E. Post-war visit to Röntgen's laboratory. AM. J. ROENTGENOL. & RAD. THERAPY, 1945, *54*, 547–552.

Fig. 3. Laboratory room in which Röntgen discovered the x-ray. By 1923 when this photograph was made, electrical conduits, outlets and motors had been added but the clock, stools and benches were used by Röntgen. At the extreme right of this photograph is the edge of the door that is pictured in Figure 4.

was my other letters from my former teachers and his old friends in the Department of Physiology at the University of Wisconsin that opened his heart and caused him to turn over his class to an assistant so that he might devote the rest of the day to me.

It was my purpose, I told von Frey, to see for myself the laboratories in which Röntgen discovered the x-ray. He escorted me to the nearby Physical Institute and introduced me to the current director, Professor Wagner, but the reception was far from cordial. Röntgen had been gone from Würzburg for twenty-three years and in the few weeks since his death his old institute had not had time to get used to the idea that now he belonged to them again rather than to Munich where the closing years of his life had been spent. Middle-class Germans and particularly university people had more pressing problems than Röntgen museums and sentimental journeys by Americans because an explosive inflation of the currency was ruining them. This inflation was generally attributed to the disastrous outcome of World War I and, by implication, to Americans, whose armies had had so much to do with that outcome. Even with my imperfect knowledge of German, I understood enough fragments of the conversation to realize that von Frey was having no easy time penetrating Wagner's reticence and disarming his conviction that this visiting American was a nuisance and probably had better be shown the door. He persevered, however, and gradually under his persistent urging and his unfailing smile, Wagner's suspicious reserve melted into helpfulness that at times became almost cordial.

At that time there were sharp restrictions

Fig. 4. Door through which Röntgen passed x-rays. It is said that Röntgen dismantled this door to determine why plates taken through it showed certain bands of decreased density. He demonstrated that these bands were shadows cast by white lead that had been used as a filler between panels and mouldings.

on the carrying of currency, cameras, binoculars, and so forth, across the Swiss-German border; and to avoid trouble I had brought in no camera. Instead it was my plan to employ a commercial photographer to make photographs of the laboratory if the authorities were willing. Wagner was agreeable to this and so off through the rain we went, von Frey and I, in a hired carriage to the photographer's shop. The photographer, we found, would be glad to oblige but he pointed out that this dark rainy day was far from ideal for indoor photography. Would it not be better, he said, to plan for the work today but actually do it later and then send the plates to me? This seemed reasonable, and so the three of us went back to the laboratory to decide with Wagner as to just what should be photographed.

There was to be one plate, of course, of the outside of the building to show the dedicatory plaque that had been attached to it in 1905 (Fig. 2); a second of the laboratory room in which the discovery had been made (Fig. 3); a third of the door through which Röntgen had passed a beam of x-rays (Fig. 4); a fourth of Röntgen's desk bearing his parents' photographs and his Nobel Prize (Fig. 5); and a fifth of some of the tubes, screens and targets that had figured in the discovery (Fig. 6). These arrangements being completed, I thanked Wagner for the privilege of visiting his laboratory and having it photographed and with von Frey took the photographer back to his shop where I paid him in United States dollars for the work that he had

Fig. 5. Desk and chair of Professor Röntgen. At the time of my visit in 1923, this desk was in a dusty storeroom in which there had begun to collect the material that in time would stock a museum. In the interval between my visit and the photographer's return the room was cleaned and its contents arranged for public exhibition. On the top shelf of the desk are photographs of Röntgen's parents and lying among the papers on the desk is his Nobel Prize certificate.

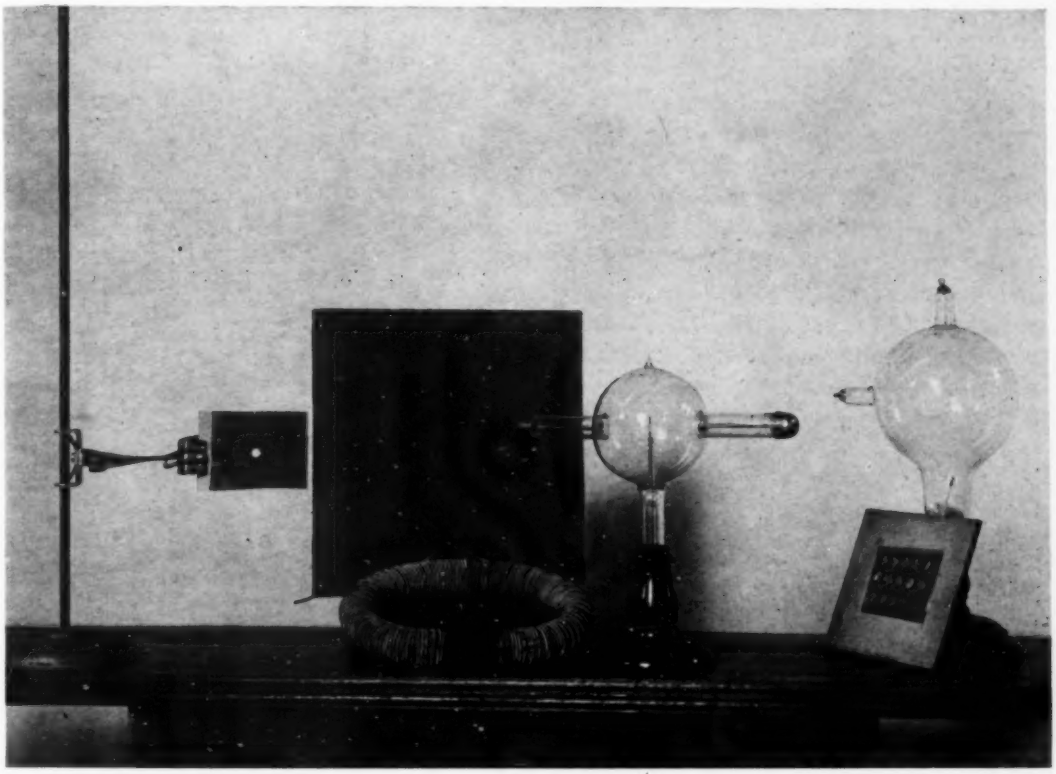

Fig. 6. Tubes, targets, screens and electromagnets used by Röntgen in connection with his discovery. Colonel Etter's Figures *3A*, *3B* and *3C* show that by 1945 much material had been added to the museum.

agreed to do on the first bright day. I had privately discussed with von Frey the propriety of my doing something for Wagner's laboratory in return for the courtesy shown me and he had suggested that the gift of a set of the photographer's prints no doubt would be acceptable. The inflation had rendered university budgets so nearly valueless that even such a modest expenditure as this could scarcely be afforded by the Institute. My payment to the photographer included a set of prints that he was to hand to Professor Wagner.

Several weeks later there arrived at my lodgings in Zurich, Switzerland, a set of prints and a letter from the photographer but no plates. The plates, he explained, could not be sent after all because of some real or imagined government regulation. The second set of prints had been turned over to Professor Wagner and please would I send the photographer another five dollars because his expenses had been greater than he had estimated.

In July, 1923, I returned to the United States and on visiting Hollis Potter in Chicago was invited by him to report at the September meeting of the American Roentgen Ray Society on roentgenology in China and on my trip to Röntgen's laboratory. It was too late to be included in the regular program but he could put me on as one of the after-dinner speakers at the dinner dance September 23. Mr. Silas W. Nourse, a professional photographer from Palisade, New Jersey, made lanterns lides for me from my Würzburg prints and was present on the evening when I showed them, having come to Chicago to assist the late Mr. Paul B. Hoeber arrange the scientific exhibit. Since I was not on the regular program, it was not required that I submit a manu-

script for publication, and being busy with other matters at the time I was glad to escape the chore. Accordingly when several friends asked where the photographs were to be reproduced and when, I explained that I was not publishing them but had authorized Mr. Nourse to make copies for anyone who wanted them.

In the years since my return to the United States in 1927 I have on occasion loaned the original prints to friends who were writing books on Röntgen and have presented sets of slides to others who are teachers of roentgenology. Recently I have seen reproductions of my photographs credited to this or that source, sometimes even with a copyright claim; and a current advertisement of one of the film manufacturers uses one of them but credits it to a Cleveland collection.

University of Chicago,
Division of Roentgenology
Chicago 37, Ill.

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Forty-eighth Annual Meeting, 1947: To be announced.

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Representatives on American Board of Radiology: Douglas Quick, New York, N. Y., B. P. Widmann, Philadelphia, Pa., F. W. O'Brien, Boston, Mass.

Committee on Arrangements: To be appointed.

Twenty-ninth Annual Meeting, 1947: To be announced.

EDITORIALS

ANOMALIES OF THE AORTIC ARCH AND GREAT VESSELS

FOR more than two hundred years there have appeared in the medical literature reports of aberrations in the development of the arch of the aorta or of arteries leading from it, and of congenital heart lesions, but it was not until the epoch-making work of Maude Abbott that the subject became of such general and widespread interest that, as Paul White says, "we no longer regard it with either disdain or awe as a mystery for the autopsy table alone to discover and to solve. She has been . . . most important . . . in establishing congenital heart disease as a living part of clinical medicine."

Maude Abbott's[1] splendid "Atlas of Congenital Cardiac Disease" was published in 1936 but we have travelled a long way since that date not so much in the basic anatomical knowledge which she so clearly elaborated but in the surgical approach to the cure or relief of some of these anomalies, particularly those of the aortic arch and its branches.

This surgical approach to the alleviation of certain of these anomalies which bring about symptoms has been made possible by the roentgenologist who has provided techniques for recognition of many of these malformations in the living subject.

Quite recently Neuhauser[2] has called attention to the importance of the roentgen diagnosis of double aortic arch and other anomalies of the great vessels. The recognition of these anomalies and their demonstration by the roentgen ray is of great importance because with the perfection of surgical technique and the mastery of the problems of preoperative and postoperative care, most of these anomalies, particularly those that are disabling or fatal in their effects, are now amenable to cure or improvement.

In Neuhauser's important paper is given a workable classification of the anomalies that involve the aorta and the vessels which stem from it. He points out that a knowledge of the embryological development of the aortic arch is essential for a complete understanding of the pathogenesis of these anomalies, but in his paper he has presented only a minimum of anomalies and he has limited his discussion to the roentgen diagnosis, emphasizing the fact that in many of these anomalous conditions the diagnosis can be made only by means of an adequate roentgenological study. He sets forth the various anomalies which are productive of symptoms and describes the detailed roentgenological examinations that are necessary to bring about a recognition of these anomalies, calling particular attention not only to the roentgenoscopic but to the roentgenographic methods of study, and above all, to the importance of outlining the esophagus with barium in order to definitely establish its relation to the aortic arch and its anomalies that might be productive of symptoms.

While some of these anomalies of development of the aortic arch may produce few or no symptoms and are frequently of only academic interest, Gross and Ware,[3] as well as Neuhauser and others, have pointed out a variety of complaints that may come from these regional malforma-

[1] Abbott, Maude E. Atlas of Congenital Cardiac Disease. American Heart Association, New York, 1936.

[2] Neuhauser, E. B. D. Roentgen diagnosis of double aortic arch and other anomalies of the great vessels. AM. J. ROENTGENOL. & RAD. THERAPY, July, 1946, 56, 1–12.

[3] Gross, R. E., and Ware, P. F. Surgical significance of aortic arch anomalies. *Surg., Gynec. & Obst.*, October, 1946, *83*, 435–448.

tions. Associated with these anomalies may be dysphagia, stridor, dyspnea, cyanosis, hoarseness, cough, pain in the upper chest and vague sensations in the arms, and in addition they may impose an extra burden on the heart because of an arteriovenous type of shunt or may lead to severe derangements in the circulation because of an obstruction in the aortic pathway. Infants and children presenting such symptoms should have careful roentgenological studies, keeping in mind the possibility of the anomalies of development of the aortic arch as a possible causative factor.

The brilliant chapter that is being written in the surgical treatment of anomalies of the aortic arch and its vessels imposes an added responsibility upon the roentgenologist in the recognition of these anomalies. Since the only possible means by which a certain diagnosis can be established is by careful roentgenological studies, it is incumbent upon the roentgenologist to fortify himself with the knowledge of the types and varieties of anomalies that may be expected and of the careful roentgenological observations and techniques which are needed in order to arrive at a proper diagnosis in these conditions which have until within recent years been considered of only academic interest but are now of vital interest because of the possibility of surgical cure.

J. Anthony Bill

ELLIS ROBERT BADER
1888–1946

DR. ELLIS ROBERT BADER, of Cincinnati, Ohio, died of chronic myelogenous leukemia on June 16, 1946, at the age of fifty-eight. Dr. Bader was born in Hamilton, Ohio, and obtained his early education there. He attended the University of Cincinnati College of Medicine from which he graduated in 1913.

During World War I, Dr. Bader served first as a medical officer in the British Army and later transferred to the United States Army. In 1919 he became associated in the private practice of radiology with Dr. William Doughty which continued

until the latter's death in 1944. He is survived by his wife, Dorothy Adams Bader, a daughter, Harriet Bader Wilks who is a registered nurse, and a son, Robert who is a medical student.

During his professional career, Dr. Bader was Radiologist at Bethesda and Deaconess Hospitals in Cincinnati, and at the time of his death, was Radiologist at Christ Hospital. He was also on the staff at the Children's Hospital. In 1940 and 1941, he was President of the staff at the Deaconess Hospital. In previous years, he had been Radiologist at St. Elizabeth's Hospital in Covington, Kentucky, and also served the Veterans' Administration as Radiologist. He had been Secretary of the Cincinnati Academy of Medicine and, from time to time, was a member of some of its committees. He was also a member of several important committees of the Ohio State Medical Association. In 1937, he was First Vice-President of the Radiological Society of North America and, prior to that, had been Counselor for Southern Ohio for many years. At the time of his death, he was Councilor for Southern Ohio of the American College of Radiology and was the Second Vice-President of the American Roentgen Ray Society. At the time of his death, he was also President of the Radiological Section of the Cincinnati Academy of Medicine. For many years, he had been Assistant Professor of Radiology at the Cincinnati College of Medicine. He was also a member of the Cincinnati Academy of Medicine, the Ohio State Medical Association and the American Medical Association.

To those who knew Dr. Bader, certainty of purpose characterized his life. He was unswervingly devoted to his home and family and after that came his professional career which he followed with fixed and definite purpose. He had an unusually wide circle of friends and acquaintances in the Cincinnati area and he was well known in national radiological circles. He was known for his honesty and integrity and had built an enviable reputation as a radiologist. In his life, he reflected only credit on the practice of medicine in general and on the specialty of radiology in particular. The American Roentgen Ray Society has lost a valuable member and his associates a devoted friend.

HAROLD G. REINEKE

EUGENE ROLLIN CORSON
1856–1946

DR. EUGENE ROLLIN CORSON died in Savannah, Georgia, on June 10, 1946, at the age of ninety. He was born in Washington, D. C., July 20, 1856. Dr. Corson was a life member of the American Roentgen Ray Society and was an honor guest at one of its annual meetings. He was also honored by invitations to present papers before learned societies in this country and abroad, and also by the Georgia Medical Society, which tendered him a testimonial dinner and meeting.

The usual obituary would be largely a repetition of a fine article entitled "A Pioneer Roentgenologist" written by Dr. E. H. Skinner and published in the American Journal of Roentgenology and Radium Therapy, 1931, Vol. 26, pp. 759–764. It is urged that this splendid tribute to Dr. Corson by Dr. Skinner be read. It well describes his life and works, and gives a bibliography of his writings.

He was a man of many interests. He read and discussed with equal fervor not only the different branches of medicine but also the allied studies of philosophy and spiritualism. He was interested in public education, especially that pertaining to the Negro, and he studied the Negro as a race. Even when his roentgenological contributions were receiving national and international acclaim, he could not be persuaded to limit his efforts to radiology. In his dramatic way he proclaimed himself a physician and surgeon and continued to follow the many calls and duties of a general practitioner. It was a sad day in his life when the radiation injury to his hand necessitated his giving up surgery and obstetrics. He was happiest when treating the humble and destitute. He was a poor business man; his income suffered. He had in his office a plaster statue of "The Charity Patient." One of my early remembrances of him was the reverent way he pointed to it and gave me some understanding of our obligations to the poor. He was artistic and temperamental and had a kind and sympathetic nature. The sufferings and hardships of others affected him, and made him an easy prey to the narcotic habitues.

It is my firm belief that had he lived in a teaching center and limited his endeavors to radiology, the stimulus of his associates and the richer clinical material would have fired his imagination to more fruitful accomplishments.

His last days were not happy. His mind was active, but his body weak. The malignant changes which followed the exposure he experienced when studying his own wrist were the cause of his death. This treatise, "The Normal Movements of the Carpal Bones and Wrists," was published in 1898.

He leaves his wife, two daughters, and a son whose grief we share.

Robert Drane

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. H. Dabney Kerr, University Hospital, Iowa City, Iowa. Annual meeting: 1947, to be announced.

American College of Radiology
Secretary, Mac F. Cahal, 20 N. Wacker Drive, Chicago 6. Annual meeting: 1947, to be announced.

Section on Radiology, American Medical Association
Secretary, Dr. U. V. Portmann, Cleveland Clinic, Cleveland, Ohio. Annual meeting: 1947, to be announced.

American Radium Society
Secretary, Dr. H. F. Hare, 605 Commonwealth Ave., Boston, Mass. Annual meeting: 1947, to be announced.

Alabama Radiological Society
Secretary, Dr. John D. Peake, Mobile Infirmary, Mobile Ala. Next meeting time and place of Alabama State Medical Association.

Arkansas Radiological Society
Secretary, Dr. Fred Hames, 511 National Bldg., Pine Bluff, Ark. Meets every three months and also at time and place of State Medical Association.

Radiological Society of North America
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting: Palmer House, Chicago, Ill., Dec. 1–6, 1946.

Radiological Section, Baltimore Medical Society
Secretary, Dr. Walter L. Kilby, Baltimore. Meets third Tuesday each month, September to May.

Section on Radiology, California Medical Association
Secretary, Dr. D. R. MacColl, 2007 Wilshire Blvd., Los Angeles 5, Calif.

Radiological Section, Connecticut Medical Society
Secretary, Dr. Max Climan, 242 Trumbull St., Hartford, Conn. Meets bi-monthly on second Thursday, at place selected by Secretary. Annual meeting in May.

Section on Radiology, Illinois State Medical Society
Secretary, Dr. H. W. Ackemann, 321 W. State St., Rockford, Ill.

Radiological Section, Los Angeles Co. Med. Assn.
Secretary, Dr. Roy W. Johnson, 1407 S. Hope St., Los Angeles, Calif. Meets on second Wednesday of each month at the County Society Building.

Radiological Section, Southern Medical Association
Secretary, Dr. Roy G. Giles, Temple, Texas.

Brooklyn Roentgen Ray Society
Secretary, Dr. A. H. Levy, 1354 Carroll St., Brooklyn 13, N. Y. Meets monthly on fourth Tuesday, October to April.

Buffalo Radiological Society
Secretary, Dr. Joseph S. Gian-Francheschi, 610 Niagara St., Buffalo, N. Y. Meets second Monday of each month except during summer months.

Chicago Roentgen Society
Secretary, Dr. T. J. Wachowski, 310 Ellis Ave., Wheaton, Ill. Meets second Thursday of each month October to April inclusive at the Palmer House.

Cincinnati Radiological Society
Secretary, Dr. Samuel Brown, 707 Race St., Cincinnati, Ohio. Meets third Tuesday of each month, October to May, inclusive.

Cleveland Radiological Society
Secretary, Dr. Carroll C. Dundon, 11311 Shaker Blvd., Cleveland, Ohio. Meetings at 6:30 P.M. on fourth Monday of each month from October to April.

Dallas-Fort Worth Roentgen Study Club
Secretary, Dr. X. R. Hyde, Medical Arts Bldg., Fort Worth, Texas. Meets in Dallas on odd months and in Fort Worth on even months, on third Monday, 7:30 P.M.

Denver Radiological Club
Secretary, Dr. W. C. Huyler, 1619 Milwaukee, Denver 6, Colo. Meets third Friday of each month at Department of Radiology, Colorado School of Medicine.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. E. R. Witwer, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Medical Society Building.

Florida Radiological Society
Secretary, Dr. J. M. Dell, Jr., 333 W. Main St., S., Gainesville, Fla. Meetings in May and November.

Georgia Radiological Society
Secretary, Dr. James J. Clark, 478 Peachtree St., Atlanta, Ga. Meets in November and at annual meeting of Medical Association of Georgia in the spring.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Illinois Radiological Society
Secretary, Dr. Wm. DeHollander, St. John's Hospital, Springfield, Ill. Meets three times a year.

Indiana Roentgen Society
Secretary, Dr. J. A. Campbell, Indiana University Hospitals, Indianapolis 7. Meets second Sunday in May.

Iowa X-Ray Club
Secretary, Dr. Arthur W. Erskine, 326 Higley Bldg., Cedar Rapids, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. Special meetings by announcement.

Kentucky Radiological Society
Secretary, Dr. W. C. Martin, 321 W. Broadway, Louisville. Meets annually in Louisville on first Saturday in Apr.

Long Island Radiological Society
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:30 P.M.

Louisiana Radiological Society
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society Meeting.

Michigan Association of Roentgenologists
Secretary, Dr. E. M. Shebesta, 1429 David Whitney Bldg., Detroit. Three meetings a year, Fall, Winter, Spring.

Milwaukee Roentgen Ray Society
Secretary, Dr. C. A. H. Fortier, 231 W. Wisconsin Ave., Milwaukee, Wis. Meets monthly on second Monday at University Club.

Minnesota Radiological Society
Secretary, Dr. Chauncey N. Borman, 802 Medical Arts Bldg., Minneapolis 2, Minn. Two meetings yearly, one at time of Minnesota State Medical Association, the other in the fall.

Nebraska Radiological Society
Secretary, Dr. D. A. Dowell, Medical Arts Bldg., Omaha, Neb. Meets third Wednesday of each month, at 6 P.M. at either Omaha or Lincoln.

* Secretaries of societies not here listed are requested to send the necessary information to the Editor.

New England Roentgen Ray Society
Secretary, Dr. George Levene, Massachusetts Memorial Hospitals, Boston, Mass. Meets monthly on third Friday, Boston Medical Library

New Hampshire Roentgen Ray Society
Secretary, Dr. A. C. Johnston, Elliott Community Hospital, Keene, N. H. Meets four to six times yearly.

Radiological Society of New Jersey
Secretary, Dr. W. H. Seward, Orange Memorial Hospital, Orange, N. J. Meets annually at time and place of State Medical Society. Mid-year meetings at place chosen by president.

New York Roentgen Society
Secretary, Dr. Ramsay Spillman, 115 East 61st St., New York City. Meets monthly on third Monday, New York Academy of Medicine, at 8:30 P.M.

North Carolina Radiological Society
Secretary, Dr. J. E. Hemphill, 323 Professional Bldg., Charlotte 2, N. C. Meets in May and October.

North Dakota Radiological Society
Secretary, Dr. L. A. Nash, St. John's Hospital, Fargo. Meetings held by announcement.

Central New York Roentgen Ra Society
Secretary, Dr. C. F. Potter, 820 S. Crouse Ave., Syracuse. Three meetings a year. January, May, November.

Ohio Radiological Society
Secretary, Dr. Henry Snow, 1061 Reibold Bldg., Dayton, Ohio. Meets during annual meeting of Ohio State Medical Association.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets first Tuesday of each month.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. J. M. Converse, 416 Pine St., Williamsport.

Philadelphia Roentgen Ray Society
Secretary, Dr. C. L. Stewart, Jefferson Hospital. Meets first Thursday of each month, October to May, at 8:00 P.M., in Thomson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. L. M. J. Freedman, 115 South Highland Ave. Meets 6:30 P.M. at Webster Hall Hotel on second Wednesday each month, October to May inclusive.

Portland Roentgen Club
Secretary, Dr. Selma Hyman, University of Oregon Medical School, Portland, Oregon. Meets monthly 2d Wednesday, 8:00 P.M., Library of University of Oregon Medical School.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Murray P. George, Strong Memorial Hospital. Meets monthly on third Monday from October to May, inclusive, 8 P.M. at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary Dr. A. M. Popma, 220 N. First St., Boise, Idaho.

St. Louis Society of Radiologists
Secretary, Dr. Edwin C. Ernst, Beaumont Medical Building, St. Louis, Mo. Meets fourth Wednesday of each month, except June, July, August, and September.

San Diego Roentgen Society
Secretary, Dr. R. F. Niehaus, 1831 Fourth Ave., San Diego, Calif. Meets monthly, first Wednesday at dinner.

San Francisco Radiological Society
Secretary, Dr. Joseph Levitin, 516 Sutter St., San Francisco 2, Calif. Meets monthly on the third Thursday at 7:45 P.M., first six months of the year at Lane Hall, Stanford University Hospital, and second six months at Toland Hall, University of California Hospital.

Shreveport Radiological Club
Secretary, Dr. R. W. Cooper, Charity Hospital, Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M., September to May inclusive.

South Carolina X-Ray Society
Secretary, Dr. T. A. Pitts, Baptist Hospital, Columbia, S. C. Meets in Charleston on first Thursday in November, also at the time and place of South Carolina State Medical Association.

Tennessee Radiological Society
Secretary, Dr. J. M. Frère, 707 Walnut St., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary, Dr. R. P. O'Bannon, 650 Fifth Ave., Fort Worth 4, Texas.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

University of Wisconsin Radiological Conference
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets every Thursday from 4:00–5:00 P.M., Room 301, Service Memorial Institute.

Utah Radiological Conference
Secretary, Dr. Henry H. Lerner, School of Medicine, University of Utah, Salt Lake City 1. Meets 1st and 3rd Thursdays monthly from 7:30 to 10 P.M., Salt Lake County General Hospital, September to June.

Utah State Radiological Society
Secretary, Dr. M. Lowry Allen, Judge Bldg., Salt Lake City 1, Utah. Meets third Wednesday in September, November, January, March and May.

Virginia Radiological Society
Secretary, Dr. E. L. Flanagan, 116 E. Franklin St., Richmond, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. Thomas Carlile, 1115 Terry St., Seattle. Meets fourth Monday each month, October through May, College Club, Seattle.

X-Ray Study Club of San Francisco
Secretary, Dr. J. M. Robinson, University of California Hospital. Meets monthly, third Thursday evening.

Cuba

Sociedad de Radiología y Fisioterapia de Cuba
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

Mexico

Sociedad Mexicana de Radiologia y Fisioterapia
General Secretary, Dr. D. P. Cossio, Marsella No. 11, Mexico, D. F. Meets first Monday of each month.

British Empire

British Institute of Radiology Incorporated with the Roentgen Society
Medical Members' meeting held monthly on third Friday at 2:30 P.M. and Ordinary Meeting at same time on following Saturday, October to May, 32 Welbeck St., London, W.1.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London.

Faculty of Radiologists
Secretary, Dr. M. H. Jupe, 23 Welbeck St., London, W.1 England.

Section of Radiology and Medical Electricity, Australasian Medical Congress
Secretary, Dr. H. M. Cutler, 139 Macquarie St., Sydney, New South Wales.

Radiological Section of the Victorian Branch of the British Medical Association
Secretary, Dr. Keith Hallam, St. George's Hospital, K.E.W., Melbourne, E. 4, Victoria, Australia. Meets monthly from March to November inclusive.

Canadian Association of Radiologists
Secretary, Dr. E. M. Crawford, 2100 Marlowe Ave., Montreal 28, P. Q.

Société Canadienne-Francaise d'Electrologie et de Radiologie Médicales
Secretary, Dr. Origéne Dufresne, 4120 Ontario St., East, Montreal, P. Q.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Radiological Section, New Zealand British Medical Association
Secretary, Dr. Colin Anderson, Invercargill, New Zealand. Meets annually.

SOUTH AMERICA

SOCIEDAD ARGENTINA DE RADIOLOGIA
Secretary, Dr. Guido Gotta, Buenos Aires, Argentina. Meetings are held monthly.

SOCIEDAD PERUANA DE RADIOLOGIA
Secretary, Dr. Julio Bedoya Paredes, Apartado, 2306 Lima, Peru. Meetings held monthly except during January, February and March, at the Asociación Médica Peruana "Daniel A. Carrión," Villalta, 218, Lima.

CONTINENTAL EUROPE

CESKOSLOVENSKÁ SPOLEČNOST PRO RÖNTGENOLOGII A RADIOLOGII V PRAZE
Secretary: MUDr. Roman Blána, Praha XII, Korunní 160, Czechoslovakia.

SOCIEDAD ESPANOLA DE RADIOLOGIA Y ELECTROLOGIA
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7. Madrid, Spain. Meets monthly in Madrid.

SOCIÉTÉ SUISSE DE RADIOLOGIE (SCHWEIZERISCHE RÖNTGEN-GESELLSCHAFT)
Secretary for French language, Dr. Babaiantz, Geneva.
Secretary for German language, Dr. Max Hopf, Effingerstrasse 49, Bern. Meets annually in different cities.

SOCIETATEA ROMANA DE RADIOLOGIE SI ELECTROLOGIE
Secretary, Dr. Oscar Meller, Str. Banual Mărăcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

ALL-RUSSIAN ROENTGEN RAY ASSOCIATION, LENINGRAD: USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

LENINGRAD ROENTGEN RAY SOCIETY
Secretaries, Drs. S. G. Simonson and G. A. Gusterin. Meets monthly, first Monday at 8 o'clock, State Institute of Roentgenology and Radiology, Leningrad.

MOSCOW ROENTGEN RAY SOCIETY
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky. Meets monthly, first Monday, 8 P.M.

SCANDINAVIAN ROENTGEN SOCIETIES
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

CANCER TEACHING DAY

A Cancer Teaching Day was held at the Hermann M. Biggs Memorial Hospital, Ithaca, New York, on October 10, 1946. The meeting was called to order at 4:00 P.M. by the Chairman, Dr. N. Stanley Lincoln, Superintendent of the Hermann M. Biggs Memorial Hospital. The opening remarks were made by Dr. Louis C. Kress, Director of the Roswell Park Memorial Institute. Two papers were given: "Cancer of the Lung" by Dr. Richmond Douglass, Senior Tuberculosis Hospital Physician, Hermann M. Biggs Memorial Hospital, and "Cancer of the Esophagus" by Dr. John H. Garlock, Attending Surgeon, Mt. Sinai Hospital, New York. The Chairman of the evening meeting was Dr. Robert H. Broad, President of the Medical Society of the County of Tompkins. The following two papers were presented: "Cancer of the Cervix" by Dr. Gray H. Twombly, Co-director, Gynecological Service, Memorial Hospital, New York, and "Cancer of the Breast" by Cushman D. Haagensen, Assistant Professor of Surgery, College of Physicians and Surgeons, New York.

ALABAMA RADIOLOGICAL SOCIETY

At the April meeting of the Alabama Medical Association, a group of radiologists met and organized the Alabama Radiological Society. The next meeting will be at the same time and place as the Alabama State Medical Association. The officers are as follows: *President*, J. A. Meadows, M.D., Birmingham; *Vice-President*, Courtney S. Stickley, M.D., Montgomery; *Secretary-Treasurer*, John Day Peake, M.D., Mobile.

CHICAGO ROENTGEN SOCIETY

At a recent meeting of the Chicago Roentgen Society, the following officers were elected for the coming year: *President*, Earl E. Barth, M.D., Chicago, Illinois; *Vice-President*, Fay H. Squire, M.D., Chicago, Illinois; *Secretary*, T. J. Wachowski, M.D., Wheaton, Illinois.

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

PEDIATRIC X-RAY DIAGNOSIS: A TEXTBOOK FOR STUDENTS AND PRACTITIONERS OF PEDIATRICS, SURGERY AND RADIOLOGY. By John Caffey, A.B., M.D., Associate Professor of Pediatrics, College of Physicians and Surgeons, Columbia University; Associate Pediatrician and Roentgenologist, Babies Hospital and Vanderbilt Clinic, New York City, etc. Cloth. Price, $12.50. Pp. 838, with 711 illustrations. Chicago: The Year Book Publishers, 1945.

In 1910, Dr. Thomas M. Rotch published "The Roentgen Ray in Pediatrics." Since then no book devoted entirely to pediatric roentgen diagnosis has appeared. The publication of Caffey's volume supplies a long standing need for a reference and textbook for both radiologists and pediatricians. In remedying this need, he has written a complete treatise, based on his experience of the past twenty years. The reproductions of the roentgenograms are all taken from the files of the roentgen departments with which he is associated. In his preface, he states his purpose is twofold: description of shadows cast by normal and morbid tissues, and clinical appraisal of roentgen findings in pediatric diagnosis. In this he has been extremely successful.

The text is divided into six sections: (1) The Head and Neck; (2) The Thorax; (3) The Abdomen and Gastrointestinal Tract; (4) The Pelvis and Genito-Urinary Tract; (5) The Extremities and (6) The Vertebral Column. In each section the normal roentgen appearance is first described followed by descriptions of the various morbid processes. The sections are well covered and throughout the reader is impressed by the wide experience of the author with a resultant authoritative expression of his clinical appraisal of the roentgen findings. The illustrations of the roentgenograms are clear and drawings are freely used to illustrate anatomical findings. The bibliography is not voluminous but is nevertheless sufficient to permit further study of any subject. The style is readable and succinct. The format is excellent.

One of the salient features of the book is the emphasis upon normal roentgen anatomy. Another is the inclusion of the most frequently occurring conditions in infants and children with omissions of those of infrequent occurrence, thus permitting more ample discussion of the former. There is little of a controversial nature in the author's opinions to which exception might be taken.

Caffey's book should be included in the library of every radiologist and as a reference book for pediatricians it cannot be excluded. For those radiologists whose pediatric patients are of limited number it is a godsend.

R. S. BROMER

ELECTRON AND NUCLEAR COUNTERS: THEORY AND USE. By Serge A. Korff, M.A., Ph.D., Associate Professor of Physics and Supervisor of Physics Research, College of Engineering, New York University. Price, $3.00. Pp. 212, with 69 illustrations. New York: D. Van Nostrand Company, Inc., 1946.

Since its introduction almost forty years ago, the Geiger counter tube has become a powerful measuring instrument for all types of radiations. Although the most startling developments of these tubes as detector and measuring instruments have taken place during the last few years and mostly in connection with atomic energy investigation, they have been used for years in numerous other research projects. In radiology, Geiger counters have been used to detect lost radium. Subsequently, surveys of protection against roentgen rays and radium were carried out with Geiger counters, and recently the instrument has become indispensable for detecting and measuring minute traces of radioactive isotopes introduced into the organism. A wide variety of counters have been built for numerous purposes, but detailed information on their construction and use is not yet generally available.

This book is welcome since it is one of the first comprehensive reports on electron and nuclear counters. It will undoubtedly shed light on many confused and haphazard ideas about counters which have been circulated during the last few years. Tracing the historical develop-

ment of Geiger counters from the Rutherford-Geiger alpha-ray counter described in 1908 and the Geiger point counter of 1913, Korff describes in great detail the theory and operation of *proportional counters* as well as self-quenching and non-self-quenching Geiger counters. This is followed by practical suggestions regarding the preparation and construction of counters and by a critical discussion of errors and corrections made in evaluating the counts. A chapter on auxiliary electronic circuits in connection with counters concludes the valuable text material. This last chapter contains a list of articles and books on electronic circuits and on counter problems as well as references to commercial sources of supply of counters, circuits and materials for their construction. This latter list, unfortunately, is far from complete.

A general bibliography as well as an author and subject index for the book is appended. To radiologists who will become more and more involved in problems requiring the use of counter equipment, this book can be highly recommended as a textbook and guide in the construction and successful operation of Geiger counter apparatus.

Otto Glasser

Ultraviolette Strahlen: Ihre Erzeugung, Messung und Anwendung in Medizin, Biologie und Technik. Von A. E. Herbert Meyer, Dr. phil., und Ernst Otto Seitz, Dr. phil. Mit einem Geleitwort von Professor Dr. B. Rajewsky, Direktor des Kaiser-Wilhelm-Institutes für Biophysik, Frankfurt a.M. Cloth. Price, $10.00. Pp. 308, with 217 illustrations and 40 tables. Berlin: Walter de Gruyter & Co., 1942. J. W. Edwards, Publisher, Ann Arbor, Michigan.

This book has been prepared by the authors for the physician, biologist, chemist and physicist who all are using ultraviolet rays in their respective fields of endeavor. So far a survey of the entire field has been lacking and this publication is presented for the purpose of filling that gap.

In an introductory chapter some of the basic principles concerning spectra and the general laws governing light waves are briefly outlined. The production of ultraviolet rays and the various types of ultraviolet lamps are discussed in detail; this chapter includes also some data regarding the sun and its spectum. Considerable space is devoted to the problem of measuring ultraviolet rays and most accepted methods, including some of their advantages and disadvantages, are described. About one-third of the book has been assigned to the physical, biological and photochemical effects of ultraviolet rays and their use. The material is organized under the following headings: Fluorescence and phosphorescence, the fundamental biologic effects of ultraviolet rays, erythema and pigmentation, biologic effects of ultraviolet rays and their application in medicine, rickets and vitamin D, therapeutic lamps and their evaluation, technical application of ultraviolet lamps. A rather interesting experiment is the use of large units of ultraviolet lamps in one of the mines of the former Krupp concern. The miners take off their working clothes in one room, pass through showers and walk naked at a set rate of speed through "radiating" gangways. It is stated that in the thus irradiated group the incidence of colds, influenza and rheumatism was decreased to about 33⅓ per cent as compared with the non-irradiated group. The bibliography contains 953 references. As a reference work covering the literature up to 1941 this book is recommended to all working with ultraviolet radiation.

Ernst A. Pohle

Physical Chemistry of Cells and Tissues. By Rudolf Höber, University of Pennsylvania School of Medicine, Philadelphia, Pa. With the collaboration of David I. Hitchcock, Yale University School of Medicine, Laboratory of Physiology, New Haven, Conn.; J. B. Bateman, Mayo Clinic, Rochester, Minn.; David R. Goddard, University of Rochester, Biological Laboratories, Rochester, N. Y.; Wallace O. Fenn, University of Rochester, School of Medicine and Dentistry, Rochester, N. Y. Cloth. Price, $9.00. Pp. 676, with 70 illustrations. Philadelphia: The Blakiston Company, 1945.

A generation of students in cell physiology has grown up under the more or less direct influence of the successive (1902–1926) editions of Professor Höber's *Physikalische Chemie der Zelle und der Gewebe;* and the present successor volume assumes its place as a valuable aid to the advanced student and investigator in the field. The subject of the book is physiology treated "as a branch of physical chemical science dealing with life as a physical, though exceedingly complex system, that may be subjected to

scientific analysis like any other natural object."

In Section 1 (91 pages), Hitchcock expounds selected aspects of physical chemistry, viz.: diffusion in liquids; reaction velocity and enzyme action; elements of thermodynamics; electromotive force; some properties of aqueous solutions. The treatment assumes that the reader already has some knowledge of the subject. Although condensed in treatment, this subject matter is competently presented. However, the necessity for this section may well be debated in view of its restricted scope and the availability of adequate texts.

In Section 2 (121 pages), Bateman presents a valuable discussion, instructive and stimulating, of large molecules, their physicochemical properties and their architectural significance in living matter.

In the next three sections (154 pages), Höber introduces the reader to the architecture of protoplasm and discusses in considerable detail the structure and permeability of the protoplast surface, and the influence of some extracellular factors on cellular activity. The wide range and complexity of the subjects treated compel admiration for the author's industry and broad background. The student will find here many facts and much else to ponder upon.

In Section 6 (74 pages), Goddard gives a comprehensive and lucid presentation of the enzymatic mechanisms underlying the respiration of cells and tissues with emphasis on the physicochemical approach. Whether it belongs in this or another section of the book, there is unfortunately no exposition of the beautifully integrated system of gaseous, and ionic equilibria in mammalian blood (and erythrocytes) as influenced by the respiratory cycle of tissues.

The subject of tissue (particularly muscle) contractility is discussed by Fenn in Section 7 (79 pages). This includes an excellent account of the pertinent experimental observations (up to September, 1942) and of the successive theories of muscle contraction.

In the final Section 8 (87 pages), Höber considers passive penetration and active transfer in animal and plant tissue, viz.: intestinal absorption; formation of urine; permeability of the body surface of animals and plants; elaboration of digestive juices. A closing discussion deals with the energetics of active transfer, the transferring devices and their mechanics.

Because of its general content and its critical approach to the problems of cell physiology, this book is almost an essential for those interested in the field.

Barnett Cohen

Karolinska Sjukhuset. Redogörelse för Första Etappen av Karolinska Institutets Nybyggnader Å Norrbacka Vid Stockholm. Pa byggnadskommitténs uppdrag utarbetad av Th. Borell, Gösta Forssell och Einar Key. Cardboard. Pp. 265, with 60 illustrations and 22 plates of plans and sections. Stockholm, Sweden: P. A. Norstedt & Söner, 1944.

Through the courtesy of Professor Gösta Forssell, of Stockholm, we have recently received a prospectus of the new Caroline Hospital of the Caroline Institute of Stockholm, and it has seemed to the Editor that the readers of the Journal wlll be glad to learn of this new institution.

The Caroline Hospital was erected by the Swedish State as a purely State institution, affiliated with the State Medical School in Stockholm, the Caroline Institute. The City of Stockholm, the County of Stockholm, and the King Gustaf V Jubileum Foundation also contributed to its construction and equipment and assist in its upkeep.

New structures built or contemplated for the Caroline Institute occupy 559,000 square meters on the outskirts of Stockholm city. The projected hospital is very elaborately described in a beautifully bound and printed book of 265 pages plus some 22 large extended drawings, showing the floor plans of the various divisions of the hospital. The institution, as projected, will have 1,056 beds, including 138 beds for the radiotherapeutic clinic. Surgical and medical outpatient departments are situated one on either side of the ground floor and the roentgen diagnostic department is one floor up.

The King Gustaf V Jubileum Clinic consists of a radiotherapeutic clinic, a radiopathological and a radiophysical department. This includes the wards, the outpatient department, therapeutic department, the offices, lecture halls, library, etc. There is also an observation ward. The radiopathological department communicates directly with the radiotherapeutic clinic. For technical reasons the radiophysical department is installed in an entirely separate building.

There is a great deal of detail in the description of the various departments. The architects'

drawings are most elaborate and contain details which would be of value to anyone interested in hospital construction.

James T. Case

Journal of the History of Medicine and Allied Sciences. January, 1946, Volume I, Number 1. Pp. 182. Published quarterly. Subscription rate, $7.50 in the U. S., Canada and Latin America; $8.50 elsewhere. Single copies, $2.50. Henry Schuman, 20 East 70th St., New York 21, N. Y.

"If a man has a sincere interest in the historical development of the knowledge of his subject, he has one of the most essential qualities of a true scholar." So said one of my colleagues, a Professor of English, in a recent informal discussion. While such a state of mind may not, to be sure, constitute all the criteria of genuine erudition, nevertheless it is a reliable single guide. As a teacher of medicine I have often pondered over the puzzling matter of why a student and many physicians will continue to use a medical term bearing a man's name, for example Thomas Hodgkin, Richard Bright or Guido Banti, day after day, without the slightest desire to ascertain who the man was, when and where he lived, and the nature of the circumstances which led to the permanent linking of his name with a certain disease state. It is difficult to comprehend why one's intellectual curiosity and depth will permit the constant use of terms and names for months or years, without manifesting some interest in their origin or historical significance.

A study of the history is not so far afield from the study of medicine. In both there is the science and the art. The scientific angle in history is compatible, at least in part, with the definition of science given by Hume, in which he says, "Science is an effectively organized body of information gained by observation, experimentation and reasoning . . . history is also . . . an art. It consists not only in collecting facts about the past, but in thinking and feeling about them. Adequate interpretation and portrayal are impossible without imagination, a sense of life, and a gift of expression." Surely some of the ways of the historian are truly valuable ones in the training of the physician.

These statements are preliminary to welcoming to medical readers a new quarterly periodical, devoted to the history of medicine and allied sciences, of which the first issue appeared on January 1, 1946. This journal, edited by Dr. George Rosen and published by Henry Schuman of New York, in its first number of 182 pages, contains 11 articles of a high scholarly standard. This issue suggests that the critical mind of the editor will select contributions for publication which are truly of a permanent rather than a fleeting value. For this he is to be commended. There has been too much medical history written in the past which is based upon a cursory study without recourse to the original sources. While such articles may have been readable, they have not always been trustworthy or of permanent value. Such efforts cannot be classified as historical research. In reality some articles contribute nothing but fragmentary summaries of existing knowledge.

No other guarantee is needed concerning the qualifications of Dr. Rosen as an editor than to know that he is the author of one of the most comprehensive, authentic, and learned medical treatises on a historical subject which has been published in recent years, namely, "The History of Miner's Disease." For this he very appropriately received the Grant Squires prize of Columbia University in 1945.

In addition to the original articles, the first issue of the new journal contains a special section "Notes and Queries" edited by Max H. Fisch, head of the Rare Book Division of the Army Medical Library. This is a unique addition and one which will undoubtedly serve as a stimulating and informative division. The book reviews in this initial issue are contributed by George Rosen, Henry R. Viets, George Urdang, and Arthuro Castiglioni. They are exceedingly well prepared and promise to be an interesting feature of future quarterly editions.

To the editor and publisher, the reviewer extends congratulations on the first issue of the new journal. It is scholarly, readable, and has all evidences of being thoughtfully and carefully planned.

C. C. Sturgis

DEPARTMENT OF TECHNIQUE

Department Editor: Robert B. Taft, M.D., B.S., M.A., 103 Rutledge Ave.
Charleston, S. C.

SIMPLIFICATION OF ROENTGENOGRAPHY BY HIGH KILOVOLTAGE TECHNIQUE*

By J. GEORGE TEPLICK, M.D.
Department of Radiology, Jefferson Medical College Hospital
PHILADELPHIA, PENNSYLVANIA

OUR investigation into roentgenographic technique began in order to shorten exposure time. With no rotating anode tube at our disposal, we could not use the short exposure times that high milliamperage allows. We were also unwilling to forego the use of a small focal spot.

To shorten the exposure time the peak kilovoltage was raised, and much to our surprise, good roentgenograms were obtained at 85 kv. (peak) for all routine Bucky work. Although our machine had a double focus tube, only the small 2.0 mm. spot was used, with 30 milliamperes. All non-Bucky screen roentgenograms were made at 75 kv. (peak), 10 ma. For 6 ft. chest films, 75 kv. (peak), 100 ma., and large (4.5 mm.) focal spot were the factors employed. Chart 1 gives all the technical factors used.

This simplified technique proved satisfactory, and had the following advantages:

1. Rapid exposures could be made without a rotating anode tube and without high milliamperage equipment, still using the small focal spot.

2. Roentgenographic technique was greatly simplified; troublesome measurements and autotransformer manipulations were eliminated.

3. The same technical factors gave good roentgenograms over a wide range of thicknesses, making unsatisfactory films uncommon.

4. The soft tissues were well visualized without underexposing the bony structures.

5. Because of the technical simplicity, re-examinations were of similar density.

6. The skin received only 10–30 per cent of the radiation usually received in roentgenography.

7. Life of the roentgen tube was undoubtedly prolonged.

The only disadvantage was the decreased contrast of the roentgenograms. However, only the "beauty" and brilliance of the roentgenogram were sacrificed; the diagnostic quality was not lowered, and frequently was enhanced.

Shorter wavelengths have been suggested for soft tissue roentgenography by some workers.[1,2,5,6] High filtration rather than high kilovoltage was employed to obtain shorter wavelengths.

At 75 and 85 kv. (peak) we were obviously employing shorter wavelength radiation. In such a beam, the number of "long" wavelengths are relatively few and do not enter significantly into the film effect.

The merit of the high peak kilovoltage technique, in the final analysis, must be measured mainly by the quality of the roentgenograms obtained. The figures in the text illustrate some of the routine roentgenograms obtained by this technique. Although of less contrast than the best conventional roentgenograms, the diagnostic

* Data from United States Public Health Service Dispensary, Washington, D.C.

Chart I

Part Examined	Distance (inches)	Kv. (peak)	Ma.	Time (seconds)	Ma-Sec.
	Non-Bucky	Screen			
Chest—posteroanterior	72	75	100	1/10–1/20	5–10
Chest—oblique	72	75	100	1/10–2/10	10–20
Chest—lateral	72	75	100	1/10–1/4	10–25
Cervical spine—anteroposterior	72	75	100	1/10	10
Cervical spine—lateral	72	75	100	1/10	10
Hand—anteroposterior—oblique—lateral	36	75	10	1/10	1
Wrist—anteroposterior—lateral	36	75	10	1/10	1
Forearm—anteroposterior—lateral	36	75	10	1/10	1
Elbow—anteroposterior—lateral	36	75	10	1/10	1
Humerus—anteroposterior—lateral	36	75	10	3/20–2/10	1.5–2
Foot—anteroposterior—oblique—lateral	36	75	10	1/10	1
Os calcis—inferosuperior	36	75	10	3/10	3
Ankle—anteroposterior—lateral	36	75	10	1/10	1
Leg—anteroposterior—lateral	36	75	10	1/10–3/20	1–1.5
Knee—anteroposterior—lateral	36	75	10	1/10–3/20	1–1.5
Thigh—anteroposterior—lateral	36	75	10	3/10	3
Chest—infants	36	75	10	1/20	0.5
Sinuses—Waters	26+ (cone)	75	10	1.0	10
Sinuses—Caldwell	26	75	10	0.9	9
Sinuses—lateral	26	75	10	0.3	3
Sinuses—base	26	75	10	1.5	15
Mastoid—anteroposterior (tips)	26	75	10	1/10	1
Mastoid—lateral	26	75	10	3/10	3
Nasal bones	26	75	10	1/10	1
	Bucky	Screen			
Anteroposterior abdomen (gastrointestinal tract, barium enema, gallbladder, pyelogram, pelvis)	36	85	30	0.5–1	15–30
Lumbar spine—anteroposterior	36	85	30	0.6–1.2	18–36
Lumbar spine—lateral (including lumbosacral joint and sacrum)	36	85	30	4–8	120–240
Dorsal spine—anteroposterior	36	85	30	0.8–1.5	24–45
Dorsal spine—lateral	36	85	30	0.8–1.2	24–36
Shoulder—anteroposterior	36	85	30	3/10	9
Skull—posteroanterior	36	85	30	0.8	24
Skull—anteroposterior	36	85	30	1.0	30
Skull—base	36	85	30	1.5	45
Skull—lateral	36	85	30	3/10	9

quality is not impaired; frequently the roentgenograms are diagnostically superior.

Sharpness is not decreased by this technique. Rapidity of exposure and the use of the small focal spot will tend to increase sharpness. Although high peak kilovoltage radiation will cause more oblique and horizontal scattering,[4] in the thicker parts the scattered components are removed by the moving grid; in the thinner parts there are not enough scattered components to make appreciable film differences. Morgan,[7] in his studies with the exposure meter, found strikingly similar roentgenograms made with the kilovoltage varying from 30 to 80 kv. (peak).

Certain advantages of the high peak kilovoltage roentgenograms are striking. The excellent soft tissue visualization is of great diagnostic value. Bones are thoroughly penetrated, with the overlapping portions clearly visible. Thinner bony structures are not "burned out." The use of

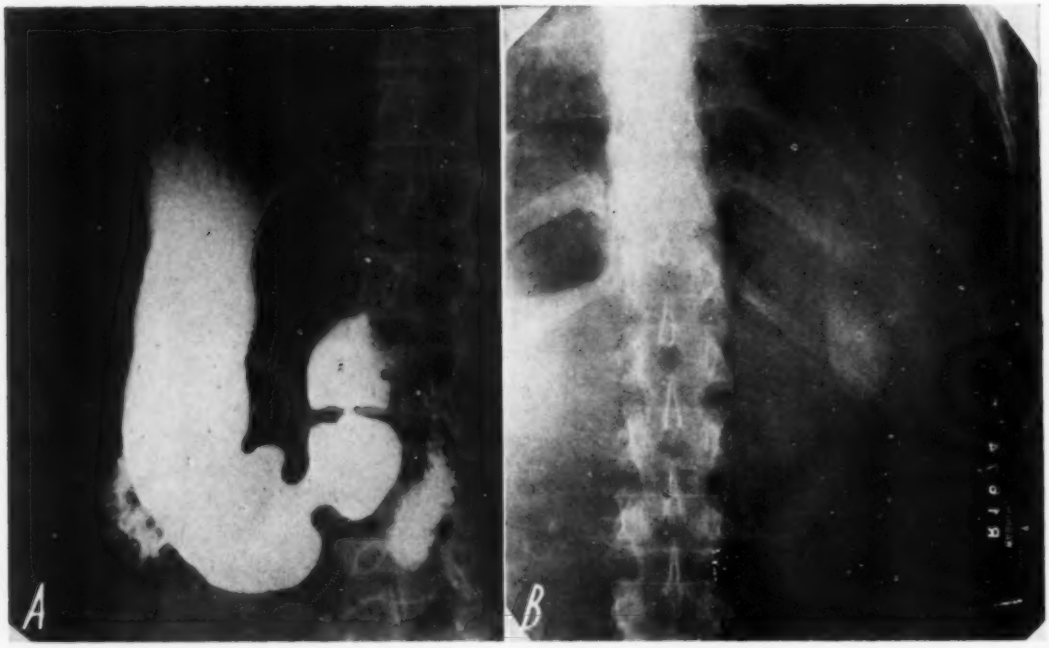


Fig. 1. *A*, gastric roentgenogram made with 85 kv. (peak), using Bucky grid. *B*, cholecystogram made at 85 kv. (peak).

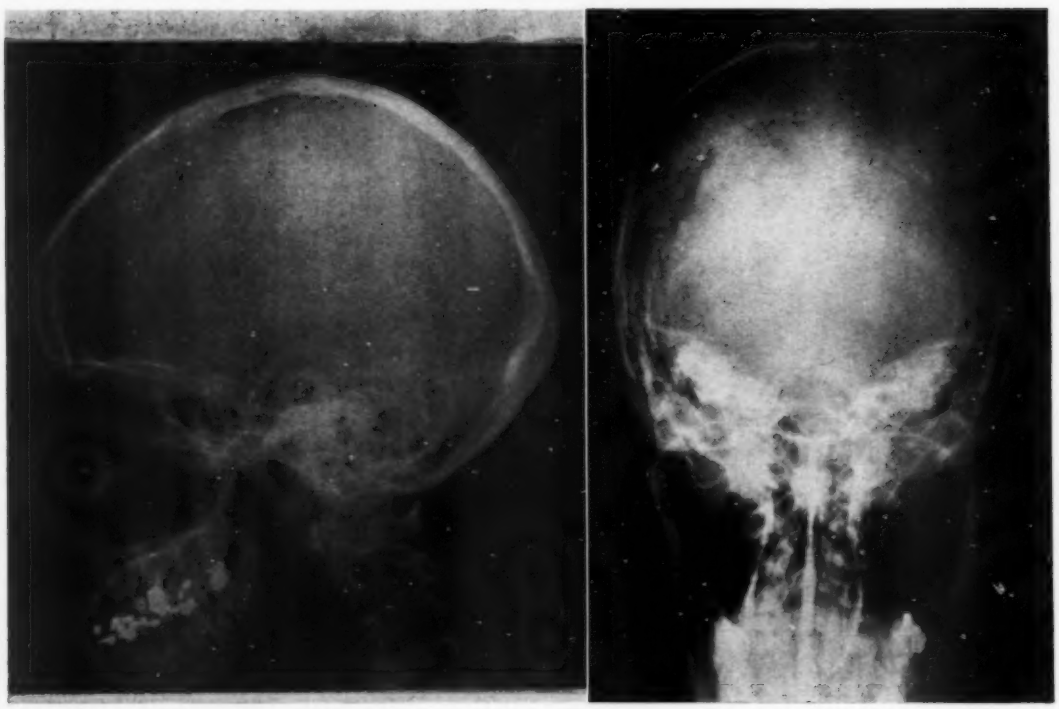


Fig. 2, Roentgenograms of the skull made at 85 kv. (peak), using Bucky grid.

the brilliant spotlight becomes unnecessary.

In the chest roentgenograms, the troublesome breast shadows become almost transparent. The lung fields are no longer brilliant below the clavicles and overcast at the peripheries. The homogeneous appearance from apices to diaphragms simplifies chest interpretation. Lesions behind the heart are exposed through the well penetrated heart shadow. Every re-examination is a remarkable replica of the previous roentgenogram.

Where extreme contrast is needed to give additional diagnostic information, a re-examination is made using a lower peak kilovoltage. The number of retakes for this reason is far less than the number of retakes when the conventional technique is used.

The high peak kilovoltage technique offers real simplification of routine roentgenography. Since only two kilovoltage settings are used, and since there is a large margin of safety, a novice technician will quickly learn routine technique and produce satisfactory roentgenograms. No measurements are necessary. Bothersome kilovolt settings, commonly a mental hazard to technicians, are eliminated. The same technical factors suffice for similar parts, regardless of moderate variations in thickness. It is remarkable that satisfactory chest roentgenograms are obtained with 75 kv. (peak), 100 ma., at 1/20 second, both for children and medium sized adults. It is also striking that for anteroposterior and lateral views of the wrist, arm, elbow, ankle leg and knee, the technical factors are iden-

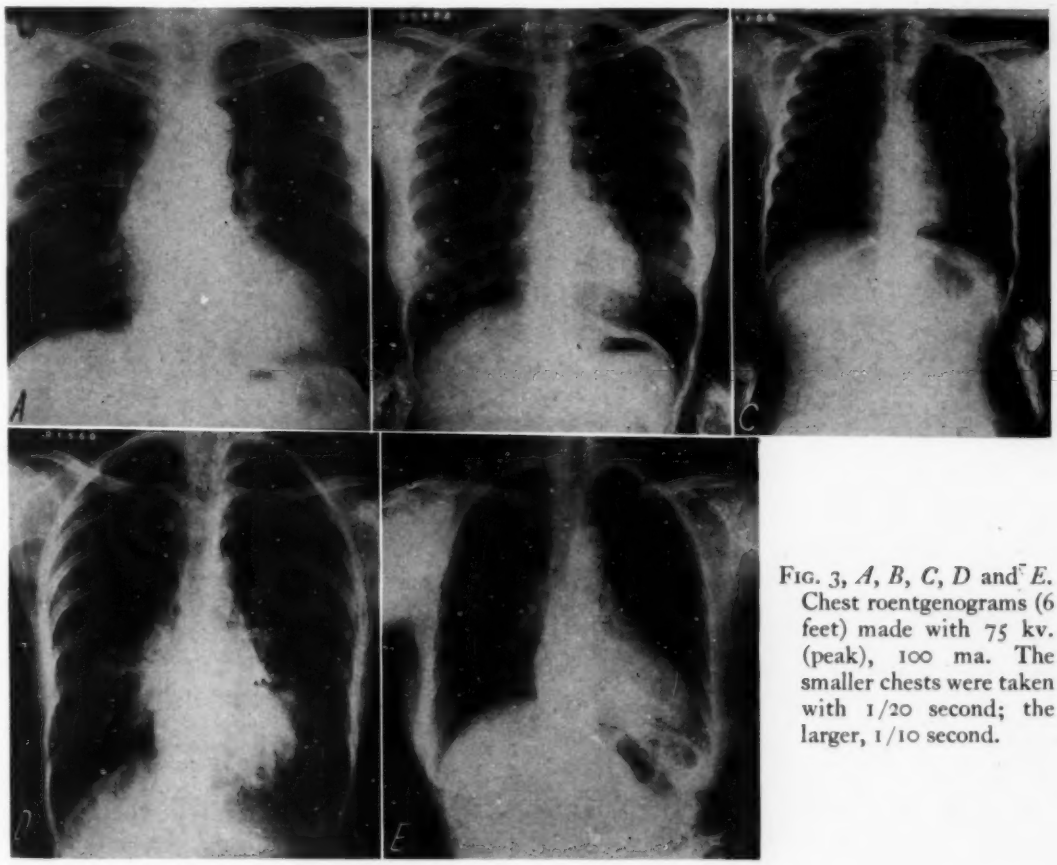


Fig. 3, *A*, *B*, *C*, *D* and *E*. Chest roentgenograms (6 feet) made with 75 kv. (peak), 100 ma. The smaller chests were taken with 1/20 second; the larger, 1/10 second.

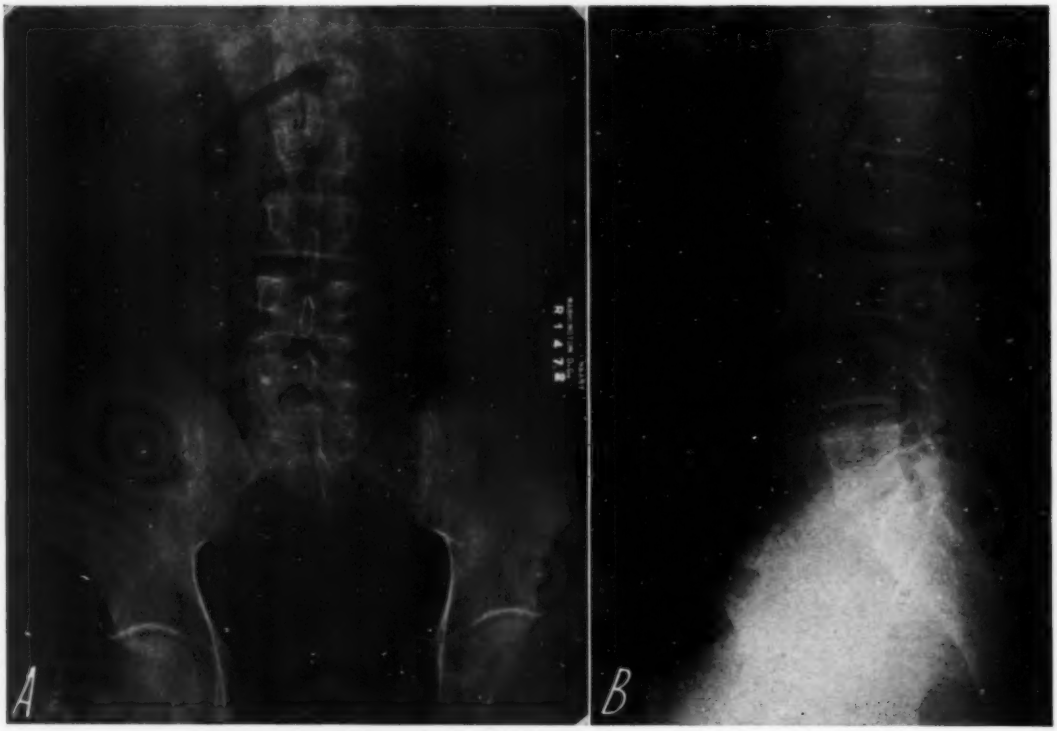


FIG. 4, *A* and *B*. Roentgenograms of the lumbar spine and pelvis made with the 85 kv. (peak) technique.

tical. It is obvious that poor exposures will be unusual, and re-examinations remarkably constant.

All exposures are relatively rapid, and the small (2.0 mm.) spot is used. With this technique, the only advantages a rotating anode tube offers are a higher milliamperage and shorter time when the Bucky grid or the 1.0 mm. spot is used. A machine powered to give up to 100 ma. is quite satisfactory.

We were greatly interested to learn that a step towards technique simplification has been made by Fuchs[3] for use in military roentgenography. A basic peak kilovoltage is set for all parts, and the milliampere-seconds are varied for thickness differences. He classifies all parts as average, small or large. The milliampere-seconds are doubled for the large and halved for the small. While this is a definite step in the right direction, there still remains a confusingly large number of peak kilovoltage settings to be remembered.

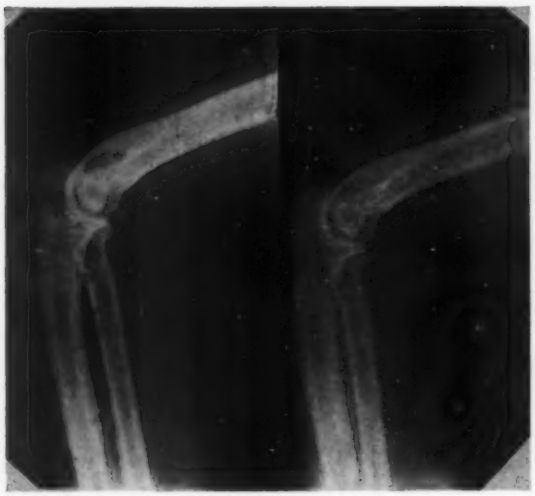
The life of the roentgen tube should be greatly increased with the high peak kilovoltage technique. For a full wave rectified apparatus, at 85 kv. (peak) and 30 ma., the maximum exposure time allowable for the 2.0 mm. spot is 20 seconds. With our

FIG. 5. Each of these roentgenograms of the extremity was made with 75 kv. (peak); 10 ma.; 1/10 second; screen at 36 inches.

Fig. 5, *Continued.*

technique, 8 seconds is the longest necessary exposure time (low lateral pregnancy film), with the majority less than 1 second. The large spot used in chest roentgenography allows 5 seconds exposure at 100 ma., 75 kv. (peak). At most (lateral chest—$\frac{1}{4}$ second) only 5 per cent of capacity is utilized.

A careful study of the roentgen output of our machine at various kilovoltages indicated that with the high peak kilovoltage technique (using no added filter), the skin



A B

FIG. 6. Lateral roentgenograms of the elbow. *A*, 48 kv. (peak); *B*, 85 kv. (peak). The films were developed simultaneously. Note the better soft tissue delineation in *B*. The decreased contrast in this roentgenogram in no way impairs its diagnostic quality.

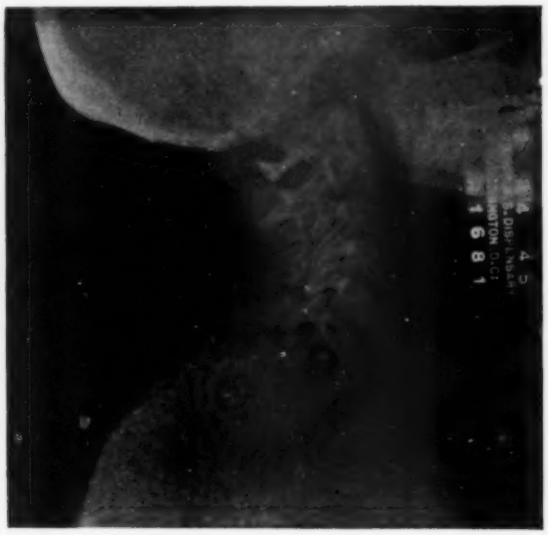


FIG. 8. Lateral roentgenogram of the cervical spine made at 6 feet using chest technique (75 kv., peak; 100 ma.; 1/20 second). Note visualization of the seventh cervical lumbar vertebra. This vertebra is consistently visualized by this technique.

dosage is reduced to 10–35 per cent of the radiation usually received in roentgenography. A complete report on this interesting and important advantage will be made in a later publication.

While the 75–85 kv. (peak) method described above proved simple and quite satisfactory, these are quite arbitrarily chosen kv. (peak) values. In the course of our experimenting, we found that a 65–75 kv. (peak) system (65 kv., peak, for non-Bucky; 75 kv., peak, for Bucky films) is also adequate and simple, giving somewhat better contrast. Certainly any fixed kv.

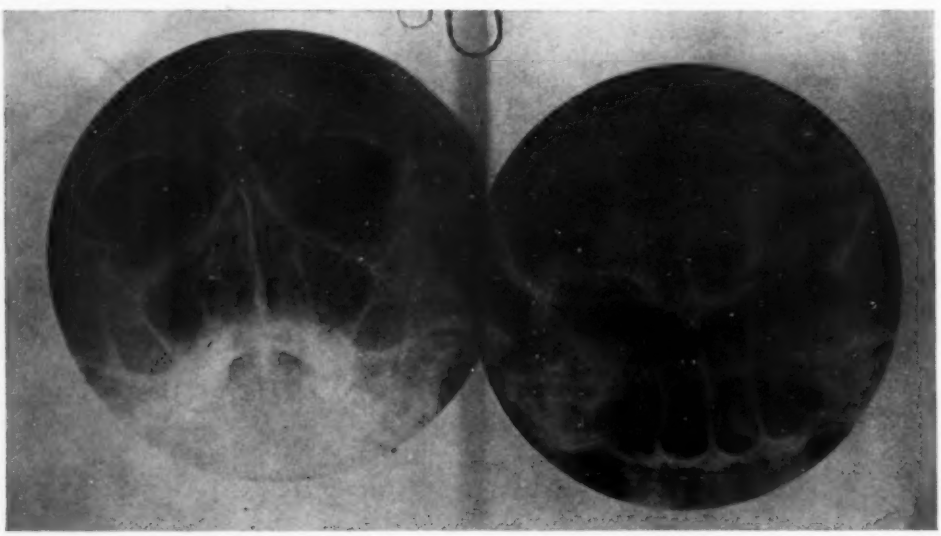


FIG. 7. Sinus roentgenograms made at 26 inches, with cone, using the 75 kv. (peak) technique.

(peak) system of roentgenography will be gratefully received by technicians, whether student or skilled veteran.

SUMMARY

A simplified system of high kilovoltage, low milliamperage technique is described. Exposures are fairly rapid, and a small (2.0 mm.) spot is used except for 6 foot chest roentgenograms. Although some contrast is sacrificed, numerous advantages are gained, namely: wider margin of safety for satisfactory exposures; marked simplification of work and time saving for the technician; good soft tissue visualization; a probable increased roentgen tube life; a markedly decreased roentgenographic skin dose.

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ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

ROENTGEN DIAGNOSIS

SKELETAL SYSTEM

JUNGMANN, H., and STERN, V. S. An unusual case of joint disease (a possible example of arthritis psoriatica). *Brit. J. Radiol.*, Dec., 1944, *17*, 383–385.

A woman of fifty-one had had sudden pain and swelling in her finger joints twenty years ago; it soon spread to other joints. Eighteen years ago she was admitted to hospital and has been bedridden since. Her limbs were drawn up so that she could not use them and she complained of severe pain all over her body and sleeplessness due to pain. She was very much emaciated with great atrophy of the muscles. Her hands and fingers were the size and appearance of those of a child of seven. There were no active or passive movements of the lower limbs. There were big irregular red patches on her skin which showed much scaling. The base of the ulna had disappeared and while the base of the radius still showed some signs of normal shape the joint space was completely obliterated. On the right side the carpal bones except the pisiform had completely disappeared; on the left some residues of the scaphoid and capitatum could be seen. The metacarpals were short and plump and tapering at the ends. Some of the phalanges had completely disappeared and the rest were tapering. The bones of the foot showed the same changes but to a lesser degree. Roentgenograms of the hands and feet are given.

Joint lesions in psoriasis were first observed in France during the first half of the last century. A number of cases have been reported in the literature in the United States. The joint changes generally occur after the skin lesions have begun but in this case and a few others the joint lesions seem to develop first. Bone destruction is generally not so severe as in this case. The author believes the two diseases are coordinated, that is that they are the result of the same agent acting on two different systems. —*Audrey G. Morgan.*

HOWORTH, M. BECKETT. Calcification of the tendon cuff of the shoulder. *Surg., Gynec. & Obst.*, April, 1945, *80*, 337–345.

Codman and others have demonstrated that the condition called subdeltoid bursitis, or periarthritis, is in reality a lesion of the tendon cuff of the shoulder. It may be a calcification, a tear, or a "tendonitis." Calcification of the tendon cuff is probably the most common cause of pain and disability at this joint.

Anatomy. The tendons of the subscapularis, supraspinatus, infraspinatus, and teres minor muscles converge and fuse with the capsule of the shoulder joint about 1 inch from its distal margin. The common tendon capsule cuff thus formed is attached to the superior margin of the tuberosities and the anatomic neck of the humerus. The subdeltoid or subacromial bursa lies upon this tendon cuff and the greater tuberosity, and is covered by the deltoid muscle, acromion process, and coraco-acromial ligament. It is a thin-walled, transparent, hemispherical pouch, averaging about 1½ inches in diameter, and lined with synovial membrane.

Pathology. The bursa and calcareous deposit were exposed at operation in 23 shoulders The bursal wall was usually thickened and opaque, edematous and vascular.

The calcareous deposit could be identified in the floor of the bursa as a white or yellowish area, often a raised wound resembling a "boil," surrounded by a reddish zone.

The degenerated tendon often was infiltrated with calcareous granules or clumps. Bare bone was exposed in the base of some of the cavities. No communication with the joint was demonstrated, and only one had ruptured into the bursa. Most of the deposits were in the tendon of the supraspinatus, but there were several in the infraspinatus and the teres minor, one in the subscapularis muscle.

Etiology. The cause of the calcification was not obvious, but it appeared that there had been first a degeneration of the tendon followed by the deposition of calcium salts. The degeneration may be the result of impaired circulation, attrition, or both. The supraspinatus tendon, with the arm at the side, makes a curve of about 40 degrees around the head of the humerus to its insertion. Thus the use of the arm at the side, with sudden, jerky, or resisted motions toward abduction or rota-

tion, constantly repeated, appears to be the cause of degeneration and calcification of the tendons of the shoulder cuff.

Course and Stages. The symptoms and signs of this disease are not proportional to the size, shape and number and location of the calcareous deposits, nor even to their type. It is probable that the most important factor in relation to the severity of symptoms and signs is the tension of the calcareous material in the substance of the tendon.

The calcareous deposits often increase, occasionally decrease in size and density over a period of months or years, but may change in a few days. Rupture of the deposit into the bursa results in a change in the location and density of the calcareous material and is usually followed by immediate relief of symptoms and absorption of the material. Occasionally the deposits in the tendons are absorbed, but most of them persist for many years unless there is a spontaneous or surgical opening.

There is usually little correlation between the clinical and roentgenographic features of the disease.

Cases. One hundred consecutive cases have been included in this series. The average age was forty-three years, the range from twenty-four to sixty-three. Only 1 patient was under thirty years, 12 past fifty years. Forty per cent were men, 60 per cent women. The right shoulder was affected in 62 per cent, the left in 38 per cent, 14 per cent being bilateral. Definite injury was mentioned in only 3 cases. Housekeeping was the most common occupation.

Symptoms and Signs. The symptoms were pain, limitation of motion, and disability. The duration of symptoms was two days to twenty years, averaging two years.

The pain was usually described as an ache in the anterior shoulder region, often radiating down the anterior border of the deltoid to its insertion. The pain overflowed into the forearm and upper shoulder girdle region in the more severe cases. It was worse at night, making it difficult to find a comfortable position in bed, especially on the affected side. The pain was worse on activity, and often affected by weather. Abduction, or putting the hand behind the head, was difficult or impossible.

The arm was usually held at the side in internal rotation. There was atrophy of the deltoid and spinatus muscles. Tenderness of various degrees was found, usually anterolaterally just above the greater tuberosity because of the more frequent involvement of the supraspinatus muscle. The tenderness was lateral to the tuberosity with involvement of the infraspinatus, posteriorly for the teres minor and anteromedially for the subscapularis.

Roentgenograms. The typical roentgenogram revealed an amorphous calcification in the angle between the humeral head, greater tuberosity, and acromion. Usually there was one main mass, once as many as five. The supraspinatus calcifications were located just above the junction of the greater tuberosity and head, whereas those of the infraspinatus and teres minor were lower and superimposed upon the tuberosity except in internal rotation; that of the subscapularis was superimposed on the joint in internal rotation, on the head in external rotation.

The roentgenogram should be made tangential to the acromiohumeral interval, and views should be made in 45 degrees, internal, and 45 and 90 degrees external rotation, as well as in the neutral position.

Treatment. Operative removal of the calcareous deposit is the surest and quickest method of relief, particularly in the chronic cases.—*Mary Frances Vastine.*

Weens, H. S. Calcification of the intervertebral discs in childhood. *J. Pediat.*, Feb., 1945, *26*, 178–188.

Reports are conflicting as to the significance of calcification of the intervertebral discs in the adult. Recent reports indicate that such calcification can exist in the absence of symptoms. From the few case reports of calcification of the intervertebral discs in childhood, it seems probable that the condition is of more pathological significance since all cases reported showed definite clinical signs and symptoms.

The author reports a case of a five year old girl whose chief complaint was marked pain in the neck and back of the head. The symptoms had been present in a mild form for two months and had been severe for five days. An injection of novocain, following a tentative diagnosis of myositis, did not relieve the symptoms. Physical examination showed hyperextension of the neck with resistance to flexion. There was a slight hyperpyrexia which disappeared two days after admission to the hospital and two days later all pain had disappeared. Laboratory studies were essentially negative. Roentgen examination revealed a dense calcification, 6 by 5 by 2 mm., in the interspace between the

bodies of the sixth and seventh cervical vertebrae, extending from the anterior portion of the disc to its mid-portion. Review of a chest film which had been made seven months prior to hospitalization showed the lesion to have been present at that time. The calcification disappeared completely within four months.

The author summarizes 5 cases of calcification of the intervertebral discs in childhood from the literature. In all cases there was pain in the back or neck, localized to the area of the spine in which calcifications could be demonstrated. The attacks lasted from two weeks to several weeks. Where the cervical discs were involved limitation of motion of the head and neck was noted. Fever accompanied the symptoms in most cases. In all cases the calcification was in the nucleus pulposus. In adults this type of calcification is usually considered to be degenerative in nature but in the cases occurring in childhood this explanation would not apply. Trauma was a possible factor in only 1 case. The limitation of the process to a single intervertebral space would tend to rule out a general metabolic disturbance. Calcification following an aseptic necrosis such as occurs in other bones is a distinct possibility. The fact that the condition occurs in the presence of fever, leukocytosis and an increased sedimentation rate supports the theory that the calcification may result from a metastatic infection. The author apparently favors this latter theory.

Weens contrasts calcification of the intervertebral discs as it occurs in children and in adults. In the former the cervical spine is usually involved while in adults the dorsal and lumbar spines are the common sites of involvement. In the second place the calcifications in adults are practically stationary whereas in children they appear and disappear rapidly. Finally, the condition is asymptomatic in adults but is accompanied by signs of infection in the child.—*R. M. Harvey.*

MOORE, ROBERT DUNHAM. Conservative management of adolescent slipping of the capital femoral epiphysis. *Surg., Gynec. & Obst.*, March, 1945, *80*, 324–332.

The methods employed, and the results in the treatment of 44 cases of adolescent epiphysiolysis in various stages of displacement are reported. Reposition of the epiphysis was not usually attempted, and was not accomplished in any case except in those in which there was recent complete separation of the epiphysis following sudden trauma. The summary and conclusions are:

1. Of 26 hips with minimal displacement of the epiphysis, good function was obtained in 88.5 per cent following immobilization and protection of the affected hip until ossification of the capital epiphyseal growth cartilage took place.

2. Good function was obtained in 47.8 per cent of 23 hips with moderate displacement of the epiphysis following the conservative plan of treatment outlined. Thirteen per cent resulted in fair function, and 21.7 per cent had poor results. In only 1 case, or 4.4 per cent of the total, could the poor result be attributed to treatment. Results in the remaining 17.5 per cent of the cases are not known.

3. Satisfactory replacement, a living epiphysis, and a normally functioning hip followed prolonged adhesive skin traction and immobilization in 1, or 33.3 per cent, of 3 cases with complete separation. In 2 cases, poor results were due to necrosis of the epiphysis.

4. Epiphyseal necrosis may occur naturally in cases with minimal displacement. It is not uncommonly present in cases with moderate displacement first seen after bony union of the epiphysis has occurred. It is frequent following complete separation of the epiphysis due to sudden trauma.

5. Death of the epiphysis, as a complication following manipulative or surgical reposition of the epiphysis, is reported in a relatively high percentage of such cases in the literature. This disturbance was not a complication in this series of cases in which adequate conservative treatment was employed.

6. The methods used in the care of these patients are in contrast to manipulative or operative measures of treatment which increase the probability of interruption of the blood supply to the epiphysis. Long term evaluation of these divergent methods, with reference to late disability from traumatic arthritis, cannot be made until follow-up studies of representative series of such cases have been made twenty to thirty years after treatment.—*Mary Frances Vastine.*

MOORE, ROBERT DUNHAM. Aseptic necrosis of the capital femoral epiphysis following adolescent epiphysiolysis. *Surg., Gynec. & Obst.*, Feb., 1945, *80*, 199–204.

Adolescent slipping of the capital femoral epiphysis is not infrequently followed by oc-

clusion of the blood supply to this epiphysis. This complication occurs as the result of damage to the blood vessels of either the visceral capsule or the ligamentum teres during the period of progressive displacement. It may follow the trauma of surgical or manipulative procedures undertaken to replace and fix the epiphysis in its normal position on the neck of the femur. Subsequent distortion of the shadow of the head, narrowing of the shadow of the articular cartilage, and irregular density of the ossification center are usually seen in the roentgenogram. These findings may be accompanied by pain, deformity, and progressive loss of motion of varying degree.

During the early growing period the articular cartilage appears to be less dependent upon the circulation of the underlying bone. With interruption of the blood supply to the ossification center, the synovia may furnish adequate nutrition to the cartilage which continues due to proliferate, and thus becomes thicker than normal as in Legg-Perthes' disease. Restoration of the circulation to the underlying bone leads to creeping replacement of the necrotic bone followed by resumption of endochondral ossification, but the shadow of the articular cartilage on the roentgenogram usually remains slightly wider than that of the normal side due to this discrepancy between growth of the cartilage and replacement of its deeper portion by bone.

Even in adolescence, near the end of the growth period, the articular cartilage may remain alive after devitalization of the underlying bone. However, the rate of growth of the cartilage at this age is much slower than that of the younger child and cessation of endochondral ossification may not lead to any appreciable increase in the depth of the articular cartilage as is seen in Legg-Perthes' disease. In the young adolescent it appears that much of the deeper bone of the cartilage may undergo degeneration and subsequently become ossified after revascularization of the underlying bone. If such degeneration involves much of the deeper portion of the cartilage, ossification of this zone may actually lead to narrowing of this structure.

Summary.

1. In the first case reported by the author, the bone of the epiphysis underwent necrosis but most of the articular cartilage remained alive. Bony union occurred, and at the end of one year about two-thirds of the dead bone had been replaced by new bone. The articular cartilage was narrowed as its deeper zone had undergone degeneration and ossification, and growth of its superficial zone and had been inadequate to preserve the normal depth of this structure.

2. In the second case there was nonunion and both ossification center and articular cartilage underwent absorption and fibrocartilaginous replacement or ossification during the process of repair. Only a small amount of the necrotic bone had been absorbed and replaced by new bone.

The acetabular cartilage in 1 case appeared to remain viable. Early degenerative and reparative changes in this structure were noted.

The synovial membrane in both cases showed chronic nonspecific inflammatory changes.—*Mary Frances Vastine.*

McElvenny, Robert T. Roentgenographic interpretation of what constitutes adequate reduction of femoral neck fractures. *Surg., Gynec. & Obst.*, Jan., 1945, *80*, 97–106.

The suggestions for standardization of what constitutes adequate reduction of intracapsular femoral neck fractures are presented. These suggestions are the result of studying numerous roentgenograms over a six year period and of attempting to find certain characteristics which were present in those roentgenograms in cases that were successful but which did not obtain in roentgenograms of cases that were not successful.

1. The method described for obtaining reduction of femoral neck fractures is based on complete relaxation under an anesthetic and then pulling the limb down in external rotation and slight abduction. While traction is on, gentle flexion and toggling of the hip is carried out until the neck passes the head. The limb is then internally rotated in its limit. No resistance should be felt in carrying out this maneuver. The patella should face directly medially and the internal femoral condyle should be directly on the small sandbag that supports the knee. When this position is obtained, the limb is abducted to neutral or slightly beyond. An anteroposterior and, if desired, a lateral film is now taken.

2. If in the anteroposterior view, the criteria are satisfied, the guide is inserted, the guide wire is put in, and anteroposterior and lateral views are now taken. If the wire is perfectly

centered in both views and the hip is reduced in both views, the nail is driven home. Anteroposterior and lateral roentgenograms check the depth and position of the nail and the reduction. If these are satisfactory, the guide and guide wire are withdrawn and the wound is closed.

3. The reduction described is applicable to the operating room and a flat wooden table with a cassette box under its top proves as satisfactory as any table,

4. All standards described in this paper are based on roentgenograms taken with the limb in full internal rotation. In this way the neck length is established. If the head and neck are separate from one another and there are no overlapping shadows where a portion of the neck covers a portion of the head. If overlap is present, it means that a complete reduction has not been obtained because if complete internal rotation is present any overlap indicates overriding or angulation, but not end-to-end contact. Any roentgen view short of complete internal rotation distorts the relationship of the head and neck and makes interpretation of the film practically worthless.

5. A satisfactory reduction is one in which the limb is in complete internal rotation and at least neutral lateral position. The neck fragment is *well under and well inside* the head fragment as shown in the anteroposterior view. In the lateral view the head and neck are in line with no angulation and no overlap. The fracture surfaces are apposing one another.

6. A satisfactory fixation is one in which the fixation material enters the shaft of the femur *at or below the level of the lesser trochanter*, runs through the lower one-half of the neck parallel to the calcar femorale, and centers the head in both planes.

7. The main purpose of the fixation is to act as a guide which allows the head to settle on the neck in a position in which the weight thrust is directly applied to the fracture site without shearing or torsion forces being exerted on the fixation material.

8. The author wishes to stress that all his failures have occurred in hips that have been imperfectly reduced. That is, the neck fragment has not been placed well under and well inside the head fragment.

9. If a hip cannot be reduced or if there is doubt as to the effectiveness of reduction, either an immediate intertrochanteric or a subtrochanteric osteotomy should be performed.—*Mary Frances Vastine.*

Haggart, G. E., Hare, Hugh F., and Marks, J. H. Clinico-pathological conference [osteochondrosarcoma]. *Radiology*, Oct., 1944, *43*, 378–382.

The subject of this conference was a man of thirty-three admitted to the Lahey Clinic September 7, 1943, for a tumor of the right thigh of two years' duration. He had been given Coley's fluid in July and 1,000 r in divided doses. From the physical and roentgen findings a diagnosis of sarcoma of undetermined type of the inner proximal right thigh was made. Operation was advised and the patient told that the tumor was probably malignant. On the advice of his local physician he refused operation. He wrote in February, 1944, saying that he was disabled on account of increasing pain and asked for admission to hospital for whatever treatment was thought advisable. He was admitted to the New England Deaconess Hospital March 28, 1944, and after a careful consideration of the dangers of radical surgery and the inevitable serious consequences without surgery, excision was decided on.

Operation was performed in two stages, the first stage being a ligation of the right hypogastric artery. In the sesond stage the ischiorectal fossa was exposed and found to be completely filled by the tumor which pressed directly against the rectum and extended downward into the thigh. It originated from the rami of the right ischium and pubis and formed a large mass 25×15 cm. in size which was encapsulated and appeared to be benign. It was removed en bloc and the muscles which had been detached to reach it were reattached. The patient was given a transfusion 500 cc. of blood during the operation. He made an uneventful recovery and the wound healed by first intention. Microscopic examination showed that the tumor was an osteochondrosarcoma.—*Audrey G. Morgan.*

BLOOD AND LYMPH SYSTEM

Corcoran, William J. Erythroblastic anemia. *Radiology*, Oct., 1944, *43*, 373–375.

Erythroblastic anemia or Mediterranean anemia is seen only in races in the Mediterranean area, particularly Greece and Italy. It is a slowly progressive anemia with a large number of nucleated red cells. All methods of treatment so far have proved ineffective though Borzell reports that one of his patients improved under roentgen treatment. The spleen is

enlarged and the patients have a mongoloid appearance. There is osteoporosis of the long bones and often pathological fractures. The space between the tables of the skull is widened and the outer table becomes so thin that it cannot be seen on the roentgenogram. The trabeculae between the inner and outer tables give the appearance of hair standing on end.

A case is described in a boy eight years of age of Italian parentage admitted to hospital in Carbondale, Pa., April 15, 1943, for a roentgen examination of the left femur. The bone was found completely fractured at the lower third. Further examination showed the skull characteristic of erythroblastic anemia. Roentgenograms of the femur and skull are given. The blood count showed 2,750,000 red cells, 14,000 leukocytes and 44 per cent hemoglobin; there were 10 to 15 nucleated red cells to the oil immersion field.

The patient left the hospital after the fracture healed. Later splenectomy was performed at another hospital. On January 3, 1944, he was alive but in poor condition.—*Audrey G. Morgan.*

GENERAL

Bell, Joseph C., and Heublein, Gilbert W. Diagnostic roentgenology in an army general hospital during the present war. *Radiology*, Nov., 1944, *43*, 425–485.

This article is a memorial lecture delivered in honor of Dr. Preston M. Hickey who was one of the pioneers in establishing radiology in the Army General Hospitals where it is now firmly established. The radiologist in the Army general hospital has an opportunity to see a wide range of cases and to follow them up as the patients are kept until all that is possible has been done for them and this often involves a treatment extending over many months. The Percy Jones General Hospital at Battle Creek, Michigan, has a well equipped roentgen department and the work there is discussed in detail. Roentgenograms are given of lesions in the various systems and regions of the body and illustrative cases described. Photographs are also given of the various types of equipment used and a map of the world given showing the various parts of the battle front from which patients have been sent to this hospital.—*Audrey G. Morgan.*

Patterson, Russel H., and Anderson, Fred M. War casualties from prolonged exposure to wet and cold. *Surg., Gynec. & Obst.*, Jan., 1945, *80*, 1–11.

The observations in this article that are of particular interest to the radiologist are included under the following headings:

Gas-Injection and Roentgenological Findings. Three patients with extensive gangrene, in whom anaerobic cultures were consistently negative, showed by roentgen examination multiple circumscribed rarefied areas in the soft tissues of the feet.

The significance of such roentgenological findings is at times doubtful as separation and retraction of dry gangrenous tissues, as well as liquefaction of tissue, will alone or in conjunction with gas-forming organisms, produce similar changes in tissue density. Such changes may also be produced by other organisms such as anaerobic streptococci, along with various aerobes, as *Streptococcus pyogenes* and *Staphylococcus pyogenes*. One differential point is that changes in tissue density which are due to retraction or liquefaction generally manifest themselves slowly and may vary little from week to week, while changes due to gas gangrene may come on more suddenly and spread more rapidly, the more severe cases being accompanied by symptoms and signs of toxicity.

All patients in which gas bacillus infection was suspected, in addition to sulfa drugs or penicillin or both, were given prophylactic roentgen treatment of 50 to 100 r twice daily for three to five days, as advised by Kelly and Dowell.

The roentgenograms revealed in most cases a cecalsification of nearly all the bones of the feet, probably due to immobilization. Where complete deep necrosis had taken place and there was loss of blood supply to the tissues, the bones were white and dense, little or no calcium being lost.

Foot Deformities. A majority of patients seen, even including those with only second degree damage, developed a foot deformity. This was generally of a claw foot type, with varying degree of pes cavus. The great toe was pulled downward into plantar deformity and the intervals between all of the toes was increased.

The mechanism of production of the rather characteristic foot deformity is probably as follows: The muscles of the foot consist of two groups—the long extrinsic ones from the leg and the short intrinsic ones within the foot. There are flexors and extensors in both groups. An intricate balance of all these muscles is

necessary for the proper functioning of the foot. It is generally recognized that on prolonged disuse, muscle atrophy is more marked in the intrinsic group than in the extrinsic group. At the time of the original exposure, chilling and anoxia of all tissues of the feet were probably greater than of the deeper tissues of the leg. This resulted in greater damage to the intrinsic muscles of the feet and their nerve and blood supply, than to corresponding structures higher in the leg. Thus the muscles of the foot were first weakened by direct damage and secondarily by atrophy of disuse with imbalance in favor of the long muscles. The long extensors to the 2nd, 3rd, 4th and 5th toes then pulled the metatarsophalangeal joints into a marked plantar flexion. The flexora to the great toe being stronger than the extensors resulted in a plantar contraction of that toe. The increased space between the toes was due to the swelling of the tissues, plus the fixation in contraction of the lumbrical and interosseous groups. Some degree of pes cavus is produced by the contracture of the muscular and fascial structures in the plantar aspect of the foot. The appearance of cavus is exaggerated, however, by atrophy of the plantar soft tissues and by the plantar deformity of the great toe.

These findings call attention to the importance of proper support during the acute phase following injury of the extremities and to early and continued active exercises to prevent or lessen permanent deformity.—*Mary Frances Vastine.*

ROENTGEN AND RADIUM THERAPY

Campbell, Paul A. Aerosinusitis—its cause, course, and treatment. *Ann. Otol., Rhin. & Laryng.*, June, 1944, *53*, 291–302.

The great increase in aerial transport and the widespread use of low pressure chambers for the indoctrination and classification of flying personnel which has occurred during the present world conflict has focused attention upon certain conditions produced by the more or less rapid changes in barometric pressure, consequent to ascent and descent. From the viewpoint of the otorhinolaryngologist, the most important of these conditions is aero-otitis media. However, another condition, aerosinusitis, is a very definite entity and may at times be quite spectacular. In the experience of the author, aerosinusitis presents an incidence of about one-twentieth that of aero-otitis media and occurs under similar conditions.

Definition. Aerosinusitis is an acute or chronic inflammation of one or more of the nasal accessory sinuses produced by a barometric pressure difference between the air or gas inside the sinus and that of the surrounding atmosphere. It is commonly characterized by congestion and inflammation of the lining structures. Pain over the area of the sinuses is usually present. Mucosal or submucosal hemorrhage may occur. The condition may at times result in temporary or permanent change in the mucous membranes lining the sinuses depending upon the amount of barotrauma involved.

Classification of Aerosinusitis. In the normal sinus with an unobstructed ostium, free flow of gas or air between the cavity of the sinus and outside environment brings about equilibrium during ascent and descent without change in structure and consequently without sensation. Two conditions, however, may alter or prevent this free exchange of the gaseous matter. They are, first, the presence of fluid, mucus, pus, or similar substances covering the ostium, and second, obstruction of the ostium by redundant tissue or anatomical deformity. The first instance represents the basic pathology for the development of non-obstructive aerosinusitis and the second the basic pathology for the development of obstructive aerosinusitis.

1. Non-obstructive Aerosinusitis. When there is fluid, pus, or mucus covering the ostium in such a manner that it may be pushed away by relatively small pressure changes, little happens on ascent as the flow is outward. On descent, however, the direction of flow is reversed and fluid, mucus, or pus may be pressed into the sinus. Usually this phenomenon takes place without pain or other sensation but it represents a mechanism by which an uninfected sinus under certain circumstances may become infected.

2. Obstructive Aerosinusitis. When the ostium is blocked by swollen or redundant tissues or anatomical deformity, the air or gaseous contents of the cavity are trapped and during altitudinal change produce a pressure, positive in ascent or negative in descent, relative to the environmental pressure.

Blockage during descent is the more common occurrence. Under such circumstances, the relative negative pressure (vacuum) must be compensated for and alteration in structure and contents follows. The degree of alteration depends upon the degree of pressure differential.

If the degree is minor, adequate compensation will be afforded by increased secretion of fluid to fill sufficient space to equalize the pressure and release the flutter valve if such a mechanism was the causative agent. This condition may be labeled (1) first degree obstructive aerosinusitis, and is characterized by only slight, if any, pain, and usually insufficient symptoms to come to the attention of the physician. (2) Second degree obstructive aerosinusitis could readily be the designation of the condition when characterized by localized or generalized swelling of the mucosa with exudation of the tissue fluid. This degree of involvement produces definite symptoms and findings. Pain and hyperesthesia over the sinus are present, and remain for some time (one to seven days) after descent. There is usually only slight, if any, fever or leukocytosis. Roentgenography demonstrates thickening of the lining membrane and possibly some clouding produced by fluid. (3) The most severe degree of obstructive aerosinusitis may be categorized as that of third degree aerosinusitis. The differential pressure necessary to produce such a condition must be relatively great—18,000 feet or more. The resultant picture is that of extensive swelling of the lining membranes, often accompanied by extravasation of blood into the cavity of the sinus or submucosal hematoma. Excruciating pain over and about the affected sinus is present. The sensation has been described by airmen to resemble a bee sting. Fever and leukocytosis are present. Roentgenography demonstrates thickened lining, clouding by fluid or blood, and at times, submucosal hematoma, or stripping of the lining membrane. Resolution, if complete, takes from seven to twenty-one days.

Sinuses Involved. The frontal sinus seems to be involved much more often than any of the other sinuses. Incidence of involvement of the maxillary sinuses offers a poor second. Four probable reasons may be given: (1) The length and structure of the nasal frontal duct offer much more opportunity for obstruction. (2) Large sinuses are more affected than the smaller sinuses. (3) The position of the ostium of the frontal sinus would lead one to expect occasional flutter valve action of the turbinates, and redundant or swollen tissue of the turbinates. (4) The frontal sinuses have no accessory openings.

Treatment. (1) Non-obstructive aerosinusitis in most instances responds to the simplest forms of treatment if any treatment is necessary (2) The obstructive types present a somewhat different problem. In this instance, the primary attack must be directed toward equalizing the pressures inside and outside the cavities. The ideal treatment would seem to be a return to the altitude at which the block was formed, correcting the obstruction, and then a slow return to ground level. This can be accomplished either in a low pressure chamber or by actual flight, and has given good results in some cases.—*Mary Frances Vastine.*

KASABACH, H. H., and DONLAN, C. P. Roentgentherapy of hemangioma of the larynx in infants. *J. Pediat.*, April, 1945, *26*, 374–378.

The authors summarize the 8 previously reported cases of hemangioma of the larynx in infants and add 2 additional cases of their own. In both of their cases the diagnosis was followed by tracheotomy and external roentgen irradiation. One case was treated with 200 kv., 50 cm. target-skin distances, 0.5 mm. copper plus 1.25 mm. aluminum filter, 150 r daily dose. The second case was treated with the same fastors except for 100 r as a daily dose. In each case a calculated tumor dose of 1,200–1,300 r was delivered and in each case the lesion disappeared completely.

These hemangiomas in infants are believed to be congenital and are said to be more common in males although the 2 cases reported in this article occurred in females. The lesions are usually upon or above the vocal cords in adults and are usually if not always subglottic in infants. The symtoms in order of frequency are obstructive dyspnea, inspiratory stridor, hoarse cry, croupy cough, blood-tinged mucus and gross hemorrhage.

Treatment recommended by the authors is preliminary low tracheotomy followed by roentgen therapy provided 1,200 r can be delivered to the lesion.—*R. M. Harvey.*

HARVEY, ROGER A., SPINDLER, HOWARD A., and DOWDY, ANDREW H. Roentgen therapy as an adjunct in the management of acute postpartum mastitis. *Surg., Gynec. & Obst.*, April, 1945, *80*, 396–403.

This report is based upon the methods used and results obtained in treating 100 patients with roentgen therapy for acute mastitis between January, 1942, and June, 1944.

Treatment.

1. Roentgen Therapy. The initial dose of roentgen therapy used in this series of patients

varied from 40 r to 100 r and was dependent on the severity of manifestations of the disease and size of the area involved. The most acutely ill patients or those with large areas of involvement were given the small initial dose, while those with milder symptoms and smaller areas of involvement were given the larger initial dose of roentgen rays.

The number of treatments used in this series varied from one to five per breast and averaged 2.3 treatments per breast. The total dosage per breast varied from 40 r to 400 r and averaged 165 r. In general if a patient does not show satisfactory response in thirty-six to forty-eight hours, during which time two or three roentgen treatments have been given, this form of therapy should be discontinued.

Patients with superficial infection only or those with small flat breasts with superficial or deep infestions, were usually treated at 120 kv. and with 3 mm. of aluminum filtration. The other patients were treated at 200 kv. and with 0.5 mm. copper and 1.0 mm. aluminum filtration.

2. Nursing. Four instances of infant diarrhea, 2 of infant stomatitis, and 1 of infant parotitis were noted shortly after the appearance of maternal mastitis in this group of patients. For this reason it is preferred that the baby not nurse from the infected breast during the acute phase of the disease. Nursing is usually interrupted from the infected breast for six to eighteen hours. If the patient does not show marked constitutional reactions, it is safe to continue nursing from the uninvolved breast.

3. Pumping the Breast. The authors delay the use of a breast pump in patients with primary areolar or periareolar infection because of the massaging action which such an area receives during the pumping procedures.

4. Drying up Lactation. Any infection which cannot be cleared in forty-eight hours so seriously interferes with the infant feeding routine that function should be terminated in both breasts.

5. Breast Binders. All patients are markedly relieved when support is furnished to the breast to prevent any motion of the inflamed area.

6. Ice Packs. Ice has an immediate numbing effect on the painful area and decreases the blood flow to the involved area with resultant decrease in absorption of toxins into the general circulation.

7. Heat. In the early stages of mastitis, local heat makes the patient much more uncomfortable.

8. Fluids. No attempt is made to limit fluid intake during the course of the acute infection, even if the breasts are being dried up at the time.

9. Sedation. Threshold tolerance for pain is so variable that no routine is maintained in this aspect of treatment. Salicylates are frequently prescribed, and combined with codeine only in extreme situations.

Summary and Conclusions

1. Roentgen therapy combined with rational symptomatic treatment of 137 breasts affected by acute postpartum mastitis resulted in 2 abscesses, 1.5 per cent, as compared with 20 per cent abscesses with other methods of treatment in use prior to this series. The 2 abscesses in the roentgen treated group occurred in patients initially treated by other methods and not referred for roentgen therapy until four and seven days after onset of the disease.

2. The duration of subjective symptoms in these patients was reduced from an average of eight days with methods of treatment other than roentgen therapy to an average of 1.8 days with roentgen therapy.

3. Application of roentgen therapy is so effective in the early stages of this disease that treatment should be started as soon as the diagnosis can be definitely made, regardless of the hour. Delay in starting this treatment decreases chances of recovery without surgery. —*Mary Frances Vastine.*

Herger, Charles C., and Sauer, Hans R. Androgen control therapy in 130 cases of carcinoma of the prostate. *Surg., Gynec. & Obst.*, Feb., 1945, *80*, 128–138.

It is the purpose of this paper to analyze the results of endrogen control treatment in 130 patients with prostatic carcinoma. Seventy-nine of these cases were followed up for one to two and a half years; 27 of the remaining 51 patients were following for a period of from four to twelve months and 24 died of the disease (16) or of other causes (8) within the first year after beginning treatment.

Analysis of Study.

1. Favorable response to this method of treatment of prostatic cancer was accomplished in numerous instances. However, during the course of prolonged observation, it became evident that a considerable number of the group of patients showing initial improvement

developed into delayed failures. Thus, the conclusion is that even the most spectacular improvement in any patient has to be viewed with skepticism as to the ultimate outcome.

2. In the treatment of patients with far advanced or metastatic disease, androgen control treatment has proved its value as a method to prolong life. The degree of response to this method of treatment is much more impressive as a rule when the poor results accomplished by external or interstitial irradiation.

3. The results obtained were most favorable in the group of patients with metastatic lymph node involvement. All but one of the patients with metastatic lymph node involvement responded with complete regression of the metastases and so far, none of them has developed recurrence.

4. In contrast, it has been the authors' experience that spread or local extension of bone metastases is not arrested as a rule by androgen control treatment. Also this method of treatment does not protect the patient with nonmetastatic cancer of the prostate from developing metastases at a future time.

5. In spite of the apparent ineffectiveness of androgen control therapy on bone metastases proper, it cannot be denied that many of these patients derive temporary benefit from this method of treatment, such as disappearance or improvement of pain, gain in weight, and increase in well-being.

6. The authors are in agreement with Nesbit and Cummings, who express the opinion "that the maximum benefit to the patient maybe derived by delaying endocrine therapy until indicated by the onset of symptoms arising from the advanced or metastatic lesions."

7. Castration should be reserved for patients with metastatic disease, patients in whom metastases are suspected, or patients in whom rapid enlargement of the primary lesion becomes apparent. It is the authors' impression that castration is the more effective procedure if quick relief from pain in the object of therapy. Combined treatment consisting in orchidectomy preceded or followed by estrogen administration does not seem to offer any advantage over castration alone.

8. Exclusive stilbestrol medication should have its place in selected cases of prostatic cancer: (1) in patients with operable carcinoma of the prostate who refuse surgery; (2) in patients who refuse orchidectomy and (3) in patients with moderately advanced lesions of apparently low grade malignancy who have few or no symptoms.

9. Failures which occur following exclusive stilbestrol administration may be improved, at least temporarily, by orchidectomy, while estrogen administered in cases of failures following castration is ineffective as a rule. It has been suggested by Huggins that castration or estrogen administration results in a reduction in the quantity and activity of circulating androgens, thereby causing decrease in size and function of the cancer cells. He also believes that immediate or delayed failures of androgen control therapy are due to the presence of extragonadal foci of androgen production, notably in the adrenal cortex.

10. With this idea of Huggins' in mind, the authors have given external roentgen irradiation to the pituitary in 23 patients (200 kv. and delivering a total of from 1,000 to 1,700 roentgens into the region of the sella turcica). Conclusions from this treatment are that irradiation of the pituitary produced neither an appreciable decrease in the serum acid phosphatase activity, nor increase in the patient's weight nor improvement in the appearance of metastatic bone lesions. However, some degree of palliation seemed to be accomplished in that the rapid retrogressive course in delayed failure patients was slowed down at least temporarily in some of the cases treated.

11. Radiation therapy of the pituitary is not a substitute for castration or estrogen administration.—*Mary Frances Vastine.*

Abel, Stuart. Androgenic therapy in malignant disease of the female genitalia; preliminary report. *Am. J. Obst. & Gynec.*, March, 1945, *49*, 327–342.

In an extensive laboratory study of gonad hormones in 1932, Moore and Price postulated their conception of hormone interactions to include four principles: (1) gonad hormones stimulate homologous accessories; (2) secretions produced by the pituitary stimulate the gonads to function both in germ cell production and in hormone secretion; (3) gonad hormones have no *direct* effect on the gonads of either the same or the opposite sex; (4) gonad hormones of either sex exert a depressing effect upon the pituitary which results in a diminished amount of the sex-stimulating factor available to the organism.

A series of patients with previously treated but progressively advancing malignancies of the female genitalia have been given testo-

sterone propionate in arbitrary dosage of 140 to 150 mg. weekly. This was done because in the light of laboratory and clinical studies, an investigation of the possible value of male hormone in the treatment of malignancies of the female genital tract seemed highly warranted. If by the administration of testosterone the depressing effect on the pituitary would diminish the amount of sex-stimulating hormone, and if such administration is without effect upon the heterologous secondary female organs, it seemed not unreasonable to relieve that retardation, even possible regression of the malignant growth might occur in a manner comparable to the effect of estrogens on prostatic growths.

Cases to which testosterone propionate was given included carcinomas of the breast, ovary, fallopian tube, uterine corpus and cervix. A preliminary report of 5 cases in detail including carcinomas of the uterine corpus and cervix is presented. These patients have been receiving treatment for ten months.

Conclusions. All of these patients showed striking improvement in morale with feeling of well-being bordering at times on euphoria.

There was definite improvement of menopausal symptoms such as nervousness, insomnia, headache, depression, and, in most cases, hot flashes.

Increased libido was consistent and marked.

With the arbitrary dosage used (140 to 150 mg. per week) masculinizing symptoms appeared in all patients and included hypertrophy of the clitoris, development of a beard, and voice change. Acneform eruptions occurred in 3 of the original 5 patients and in many of the subsequent cases.

Vaginal smears tended toward the atrophic state and changed little during treatment.

To date, there has been nothing to indicate any regression or even retardation of the malignant process in manner comparable to the effect obtained in estrogenic treatment of prostatic carcinomas. In one of the original patients and in two subsequent cases, metastatic lesions were seen to progress during the course of treatment.—*Mary Frances Vastine.*

Cantril, Simeon T., and Buschke, Franz. Roentgen therapy in gas bacillus infection. *Radiology*, Oct., 1944, *43*, 333–345.

Nine cases of gas bacillus infection treated with surgery and roentgen irradiation are described and illustrated with photographs, roentgenograms and fever charts. A table shows the details of treatment and results; the roentgen treatment varies as greatly from case to case as does the surgical treatment. This includes all the cases of gas bacillus infection which the authors have treated by roentgen irradiation. In all except 2 cases there was bacteriologic proof that the infection was caused by a gas-producing organism. There was no mortality but this record could probably not be maintained in a large series of cases. However Kelly reports a mortality of only 11.2 per cent in 125 cases which is much better than the record with any other form of treatment.

Gas bacillus antitoxin was given in 4 cases; the authors believe that these patients would have recovered as well without it. This opinion is supported by Kelly's statistics who had a mortality of 35 to 50 per cent in cases treated with serum therapy and surgery and of 4.3 per cent in cases treated with surgery and roentgen irradiation without serum. Nor did the cases in which sulfonamides were given offer any evidence that chemotherapy played any part in controlling the progression of the gas bacillus infection.

It is believed that a thoughtful combination of roentgen treatment and surgery will give better results than any other known treatment of this disease.—*Audrey G. Morgan.*

Sigel, Harry. Direct current combined with x-ray therapy; case of Kaposi sarcoma thus treated. *Radiology*, Oct., 1944, *43*, 386–390.

The author reports a case in which he used the direct current in combination with roentgen treatment to increase the effectiveness of the latter. The patient was a man of sixty-seven admitted because of edema of the left leg and the bluish tumors characteristic or hemorrhagic sarcoma on various parts of the body.

The roentgen treatment was given with 120 kv., 5 ma., 4 mm. aluminum filter and 25 cm. distance with an output of 15 r per minute. Each area was given a total of 1,500 r. For the electrical treatment the positive electrode, large enough to cover the whole tumor area, was placed over the tumor and the negative electrode over a nearby area so that the lines of force would travel as directly as possible through the tumor and produce the greatest possible current density at the site of the tumor. Direct current was given first for about ten minutes, then roentgen irradiation begun while the current was still on and both con-

tinued until the desired roentgen-ray dose had been given. Two areas on the arm were selected and one given the combined treatment and the other roentgen treatment alone. Though these tumors are radiosensitive it was possible to detect a markedly better response to the combined treatment than to the roentgen treatment alone.—*Audrey G. Morgan.*

Siddall, R. S. Carcinoma of the vaginal wall in a girl of fourteen; radiation therapy. *Harper Hosp. Bull.*, Dec., 1944, *2*, 54–56.

A girl came for treatment May 9, 1942, two weeks before her fourteenth birthday. She had had hemorrhage at approximately regular periods for eighteen months which was thought to be menstruation, but recently the bleeding had become almost continuous. Examination showed a soft irregular mass in the region of the cervix; a small piece of tissue examined microscopically showed advanced medullary squamous cell carcinoma but examination under general anesthesia showed that the growth was on the anterior and right lateral walls of the vagina and did not involve the cervix.

On May 22, 1942, 100 mg. radium in suitable containers and screened with 1 mm. brass and 1 mm. hard rubber was placed in the vagina opposite the tumor; the dose was 3,600 mg-hr. From May 25 to 28 supervoltage roentgen therapy was given externally. Anterior and posterior fields were used, cross-firing the pelvis. The quality of the rays was that obtained with 500 kv., and a filter of 7 mm. copper, 3 mm. aluminum and 3 mm. celluloid. The total dose per field was 600 r in air. A second series of roentgen therapy with the same technique was given from August 14 to 18, 1942, and on October 15, 50 mg. radium was applied to a total dose of 1,800 mg-hr.

The tumor disappeared in three months after the first two series of treatments. But two months later it recurred and on the third series of treatments it again disappeared. On October 25, 1944, there was still no sign of recurrence but the time is too short to pronounce it a permanent cure.

The vagina contracted so that the finger can only be introduced for about 6 cm. In spite of the heavy irradiation pubic hair and other secondary sexual characteristics have appeared. There has been no sign of menstruation since early in the treatment.—*Audrey G. Morgan.*

Kobak, Alfred J., Fitzferald, J. E., Freda, Vincent C., and Rudolph, Louis. Carcinoma of the cervix and pregnancy. *Am. J. Obst. & Gynec.*, March, 1945, *49*, 307–326.

In the past five years, the authors have observed 8 cases of carcinoma of the cervix occurring during pregnancy on the obstetric services of Cook County Hospital. These cases were thoroughly studied clinically.

Summary and Conclusions.

1. The uneven distribution of these cases whereby 6 were noted in one year would render untenable a statistical evaluation of its incidence.

2. Many white and negro patients are seen annually in this hospital and it appears that carcinoma of the cervix, at least during pregnancy, might be more frequent in white women.

3. In 7 patients the malignancy was a stratified squamous cell carcinoma and in 1 patient, who died undelivered it was an adenocarcinoma.

4. There is a tendency to regard carcinoma of the cervix during pregnancy as so improbable that other causes of bleeding are given stubborn precedence in diagnosis and therapy. The malignancy is thus diagnosed more often toward the end of pregnancy, when the growth has achieved large proportions.

5. Carcinoma of the cervix, contrary to the opinion of several contributors, continues to develop at a rapid pace during pregnancy. The earlier the diagnosis is established the sooner effective therapy may be instituted, and the better the ultimate prognosis.

6. The treatment of these cases depends upon the disposition of the pregnancy. In the first trimester the pregnancy is disregarded and deep roentgen therapy is instituted. With the death of the fetus, radium is added to the treatment. In advanced pregnancy, there is added concern for the cervix. The latter should be spared of the injuries that occur during parturition. If there is no infection as noted by the temperature or character of the vaginal discharge, a classical cesarean section is performed to spare the cervix of trauma and to leave intact the fundal portion of the uterus to facilitate the radium therapy, which is started after uterine involution has occurred. A total of 4,500 mg-hr. of radium is given in three equally divided doses. As soon after the delivery of the baby as possible, deep roentgen treatments are started,

and continued for a long period of time. In the presence of local infection by criteria mentioned, a Porro cesarean section is preferred. In pregnancies close to viability the cesarean section may be deferred, and 1,500 to 3,000 mg-hr. of radium may be given to the cervix to temporarily inhibit the progress of the new growth.

7. The ultimate prognosis for the patient hinges on the persistent continuation of the deep roentgen and radium therapy that is instituted after the disposition of the pregnancy. —*Mary Frances Vastine.*

MISCELLANEOUS

Zintheo, Clarence J., Jr. Simple fluoroscopic method of foreign body localization. *Radiology*, Oct., 1944, *43*, 376–377.

Most methods of roentgenoscopic localization of foreign bodies require special apparatus or attachments of some preliminary calibration. A method is described and illustrated which can be carried out simply using only the materials ordinarily at hand in any laboratory and without any calibration. It consists essentially of locating with the movable blade of a caliper a point alongside the patient which is in the same horizontal plane as the foreign body; it is based on the fact that shadows of objects in the same plane are displaced to an equal extent when the fluoroscope is moved. An accuracy of 1 cm. or even 0.5 cm. can be achieved easily. Directions are given for protecting examiner and patient from undue exposure to the rays and the necessity for such protection is emphasized.—*Audrey G. Morgan.*

Spear, F. G. The action of neutrons on bacteria. *Brit. J. Radiol.*, Nov., 1944, *17*, 348–351.

Experiments are described which were made to determine the lethal action of a neutron beam on colon bacilli and on spores of *Bacillus mesentericus* when they were exposed to gradually increasing doses of radiation and to compare the results with those obtained with other types of radiation. The work was done with Lawrence's 37-inch cyclotron at the Radiation Laboratory, Berkeley, California. A diagram is given showing the position of the bacterial suspensions 7.0 cm. from the target of the cyclotron and two tables, the first showing the complete details of a single experiment and the second a summary of all the experimental results.

An n-unit was used for measuring the neutron radiation; it is the neutron dose given when the Victoreen dose meter, calibrated for x-rays records roentgen. Survival curves are given for the cultures of bacteria which are exponential in form. A tentative comparison between the effects of roentgen, gamma and neutron irradiation may be obtained by determining the dose required to produce a 5 per cent lethal action and expressing the result as an r/n ratio, r being the measurement of x or gamma radiation in roentgens and n that of the neutron dose in the author's unit. The r/n ratio for colon bacilli is 3.2 and that for *Bacillus mesentericus* 5.3. The r/n ratio shows wide variation according to the material irradiated. It varies from 1 to 10 with a value of 5 to 6 in most experiments. Until the dosimetry of neutrons has been studied more completely, however, the values of the ratio must be considered provisional. It must not be assumed, however, that for equal energy absorption neutrons produce a greater effect than roentgen or gamma irradiation. It seems probable that the energy absorbed per unit volume of tissue exposed to 1 n of neutrons is about the same as that from 2.5 r of gamma irradiation.—*Audrey G. Morgan.*

Spear, F. G., and Tansley, Katharine. The action of neutrons on the developing rat retina. *Brit. J. Radiol.*, Dec., 1944, *17*, 374–379.

It has been found that degeneration in tissues irradiated with roentgen and radium rays depends on mitotic activity. The experiments here described were made to determine whether neutron irradiation at doses which permit ultimate recovery of the tissues has the same biological effect.

The irradiations were given with the cyclotron on immature rat retinas. The technique is described. A table is given showing the mitotic phase and degenerate cell counts after each dose of neutron and a similar table is given for radium irradiation. Degenerate cells appeared after one to three hours. In all the experiments this initial decrease was followed by a renewal of mitosis and with doses above 5 n this return of mitosis was characterized by a marked distortion of the phase ratio. (Doses from 2.5 n to 60 n were used.)

As the dose is increased to 60 n the initial decrease of mitosis increases and recovery is more delayed. Some recovery follows the initial

fall of mitotic rate caused by neutron irradiation but the increase to a supernormal level seen with roentgen and radium irradiation does not occur after a dose of more than 2.5 n. Up to 15 n there is an increase in the prophase count but not in the metaphase and telaphase counts.

It seems that the mitotic curves after neutron irradiation do not differ materially from those after roentgen and radium irradiation but the effect on cell degeneration is much more marked. The degenerate cell count on neutron irradiation is made up partly of cells killed outright and partly of cells that are injured and die later when they attempt mitosis. The curve of cell degeneration which accompanies increased dosage occurs much sooner and is markedly steeper than with radium. The curves given show that roughly neutrons are 2.6 times as effective as radium rays per unit energy and 6.5 times as active per n unit.—*Audrey G. Morgan.*

Halberstaedter, L. Effects of x-rays on erythrocytes irradiated in vitro. *Nature*, June 3, 1944, *153*, 683.

The author, working in the Cancer Laboratories of the Hebrew University, Jerusalem, found that irradiation of erythrocytes in vitro with roentgen rays causes hemolysis. But if a dose of 2,000,000 r is given to an erythrocyte concentration of 0.1 per cent or less in normal saline a state of resistance to hemolysis develops.

In this article experiments are described which were made to determine whether the erythrocytes rendered resistant to roentgen rays in this way are also resistant to other hemolysins such as sunlight and saponin. Human or rabbit erythrocytes were irradiated with 2,000,000 r and after twenty-four hours there was no hemolysis. After addition of eosin to the irradiated drops and non-irradiated control drops they were exposed to sunlight. Hemolysis occurred in the control drops after ten to twenty minutes but no hemolysis took place in the irradiated drops after one to two hours. Similar irradiated and control drops were treated with different concentrations of saponin; hemolysis took place in the control drops after about five minutes in a 1:15,000 saponin solution while none took place in the irradiated drops after twenty-four hours in a solution of 1:3000 saponin.

These experiments show that exposure to roentgen rays may cause complete hemolysis of erythrocytes or may render them resistant to the hemolytic action of roentgen rays, sunlight or saponin. Which effect takes place depends on the concentration of erythrocytes in saline solution and the dosage of roentgen rays. Resistance to hemolysis does not occur if serum is added to the normal saline or if the erythrocytes are irradiated in glucose solution. —*Audrey G. Morgan.*

Bisgard, J. Dewey, Hunt, Howard B., and Dickinson, R. Hugh. Effect of x-ray irradiation upon bacterial toxemia in rabbits. *Radiology*, Oct., 1944, *43*, 330–332.

Previous experiments have shown that irradiation of the abdomen in rabbits produces nonspecific antitoxin. The experiments described in this article were made to determine whether the same effect is produced by irradiation of other parts of the body. One-half the animals were irradiated over the chest and the other half over the extremities at 140 kv., 15 ma, target-skin distance, 50 cm., an inherent filter of 3 mm. aluminum and an added filter of 0.5 mm. copper and 1 mm. aluminum. It was found that antitoxin is produced no matter what part of the body is irradiated.

Further experiments showed that 20 r is the approximate minimal effective dose for rabbits and this dose appeared to produce as much antitoxin as larger doses.

Injections of serum and plasma afforded some degree of protection against *E. coli* toxin, but normal non-irradiated serum and plasma was as effective as that that had been irradiated. It is probable that the effect was due to the simple addition of the antitoxin contained in the blood and plasma to that already in the tissues of the animals. Apparently the antitoxin demonstrable in the plasma after irradiation is produced by tissues other than the blood.—*Audrey G. Morgan.*